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S *Medicine*

Medical Mission to Nepal

H. Phil Gross, M.D.*

ABSTRACT

The role of the doctor serving a mission hospital in a Third World country is described. The author had the experience of working four months at the Shanta Bahawan Hospital in Kathmandu, Nepal.

The hospital is under the auspices of the United Mission to Nepal, an umbrella group comprised of 35 different major denominations from 15 countries. It is an old converted palace, and lacks many of the amenities we take for granted in our modern hospitals.

Infectious disease accounted for a large share of

For a period of four months in the fall of 1981, I had the opportunity to serve as a Volunteer in Mission at the Shanta Bhawan Hospital in Kathmandu, Nepal. This hospital was founded by Bethel Fleming, a mission doctor in Woodstock School in India. Her husband, the principal at Woodstock School, was an avid ornithologist interested in the many species of birds in Nepal.

Bob and Bethel Fleming, along with Dr. Carl Friedrichs, made several birding trips into Nepal in the late 1940s. As a result of these trips they realized the health needs of the country. They applied for permission to establish a mission hospital in Nepal, and were granted this permission in 1952, when the country was opened to the outside world. Dr. Carl Friedrichs established his hospital in Tansen, and Dr. Bethel Fleming established hers in Kathmandu.

Both these institutions were under the auspices of the United Mission to Nepal. This is an umbrella protestant church mission, comprised of thirty-five different major denominations from fifteen different countries. As a result, I found myself working next

the case load, due to poor nutrition and lack of sanitation. The usual fractures and congenital malformations were also present, but usually seen in advanced stages due to lack of immediate medical care. The primitive communication and transportation systems contributed to the delay in care.

Treatment was dictated largely by the material available, the cost, the safety, and the necessary aftercare. The physician had to be adaptable to fit in with the local culture and traditions in order to be effective.

to Mennonites from Canada, Presbyterians from the United States, Baptists from Great Britain, Lutherans from Sweden and Norway, and Anglicans from Australia.

Nepal is a nation slightly larger but shaped much the same as Tennessee. It's topography is from 500 feet above sea level in the south, to the top of the world in the north (Mt. Everest 29,028 feet). It has fourteen million people of which ninety percent survive by subsistance farming. There are 6.4 live births per family and the population is growing at the rate of 2.3% yearly, rapidly outstripping their ability to feed themselves. There is a fifty percent mortality rate between the ages of one and ten. The reason for this is poor nutrition plus infectious disease of the gastrointestinal and pulmonary systems. The infectious diseases are due to a lack of public sanitation in the country. The life expectancy is forty-three years of age. Nineteen percent can read and write.

The hospital founded by Dr. Bethel Fleming is an old Rana palace. The name Shanta Bhawan means palace of peace. With the overthrow of the Rana dynasty by the Shah family in 1952, there were over fifty of these palaces in the valley. They have been converted for various industrial uses.

^{*}Orthopaedic Surgeon, Orthopaedic Associates Ltd., Sioux Falls, SD

Shanta Bhawan hospital has a capacity of 150 beds. Although it has three stories, there are no elevators and all patient transfer from floor to floor had to be accomplished by manually carrying them up or down. There is no heat in the building and during the winter months small kerosene burners had to be used. Running water was available in all the nursing stations. This usually came from a reservoir on the top of the building, filled with an electric pump.

Electricity supply was eratic. It was available about twelve hours each day, but never on any scheduled hour. We did have our own emergency supply, but that always took five to ten minutes to start, so during that time you continued your operation with a nurse holding a flashlight. The emergency system supplied only the main operating room lights and the nurses station. Many of the wards did not have outlets, making portable x-rays impossible.

Two operating rooms were available. Anesthesia was provided by an anesthetist from Great Britain. Due to cost and other local factors, ether was the main anesthetic used. A machine to mix ether and air has been devised at Oxford University for use in developing countries. It eliminates the need for oxygen which is very expensive and of questionable purity. Such was the case of many medicines, due to lack of quality control.

One of the most common orthopaedic diseases seen was osteomyelitis. Staphylococcus was the primary causative organism with the tubercle bacilus as a close second. Treatment consisted of wide open drainage as practiced by Sir Winnett Orr. Our modern methods of closed suction were entirely impractical due to a lack of steady electricity supply, thereby negating the suction apparatus.

Tuberculosis affected all areas of the body, but was most dramatic in the spine. The patient pictured had a paraspinal abscess, plus an old collapse of two vertebra producing the kyphosis. He was being treated with drugs and surgical drainage (Figure 1).



Figure 1

Abscesses were also seen secondary to amoeba. They could cause huge areas of tissue necrosis as seen in the next illustration (Figure 2).



Figure 2

As with all forms of care, the treatment plan had to be devised to provide a minimum amount of aftercare. The next of kin were largely responsible for the daily care of the patient, with the nurses acting only in a supervisory role. This was one of the frustrations of surgical practice in Nepal, in that there was little aftercare available and a cure had to be affected almost entirely at the operating table.

Fractures assumed an unusual epidemiologic pattern. The supracondylar fracture of the elbow was the most common, followed by fractures of the femur. This was due to a fall from considerable heights. The children might fall from a roof top or porch while flying a kite, or from a tree while gathering fodder for their animals.

Treatment was again compromised due to lack of a steady electrical (and thereby x-ray) source. The fractures were most often reduced, casted, and only later x-rayed. It was surprising how well they did.

Congenital deformities seen were those compatible with life without too much medical care. Club feet were common, (Figure 3) but myelomeningocele and severe cerebral palsy were never seen. The club foot usually presented late for treatment, and had to be managed operatively. Transportation often involved a seven to ten days' walk, so the thought of frequent outpatient visits had to be abandoned.

Burn contractures were common. Most Nepalis both cook their food and heat their homes with an open wood fire. It is not difficult to imagine the toddler falling into the fire and sustaining severe burns. If he survives, the contractures provide formidable problems (Figure 4).

Bone tumors often presented late in their evolution, and therefore dramatic in their appearance. Nowhere is the pragmatism of the Nepalis better demonstrated than in malignant tumors. Oft times,



Figure 3



Figure 4

if they could not be offered a cure, they would rather go home and die, than undergo an amputation which would render them unable to work and lead a life as a beggar.

Diseases not seen were as interesting as those observed. A case of slipped capital femoral epiphysis has not been seen at the hospital in Kathmandu. This is probably explained by the lack of nutrition and slow growth. The other "not seen" diagnosis

was that of bunions and can be readily explained by the fact that people do not wear shoes.

Although I had the opportunity to join hands with many devoted workers who have given their lives to the mission, one person stands out. Ashok Banskota was the consultant to the hospital in orthopaedics. He was a Hindu, born and raised in the Kathmandu Valley. He was educated by the Jesuit brothers in a mission school. From there he went to Delhi for his M.D. degree, and then spent six years at King's County and the affiliated hospitals in New York doing his internship and residency. So he was my bridge from the western to the eastern culture. He was my ears and mouth, and I was an extra pair of hands for him. We worked well together and developed a great deal of respect, and love, for each other.

The demands on the mission doctor and his family are varied. The doctor has to be versatile, adaptable, and ready to adjust to each new situation. He will find his treatment plan is often dictated by the local customs and resources available. In addition, if he were to take his wife and family along, the most adjustment will have to be made by this segment of the team. The cultural shock can be quite cruel and if the spouse and children cannot adjust, it would be a painful experience.

So before embarking on an adventure of this type, self-assessment is necessary. It has been one of the most rewarding ventures I have ever done. It is a time for reflection on the past, and planning for the future. And this can all be done while giving of your talents to others.

Summary

The experience of working as a mission doctor in a Third World country (Nepal) is described. Infectious diseases (osteomyelitis) of the skeletal system were very common. Congenital deformities and bone tumors were often seen in their most advanced stages. Medical care was often dictated by the local customs and resources available.

Advice is given on preparation of anyone planning this type of medical service.



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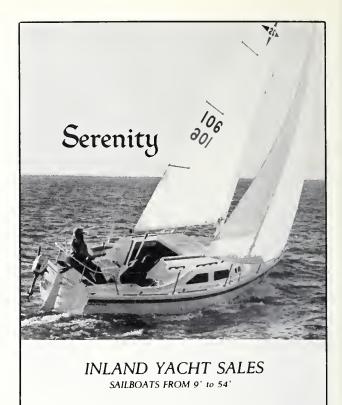
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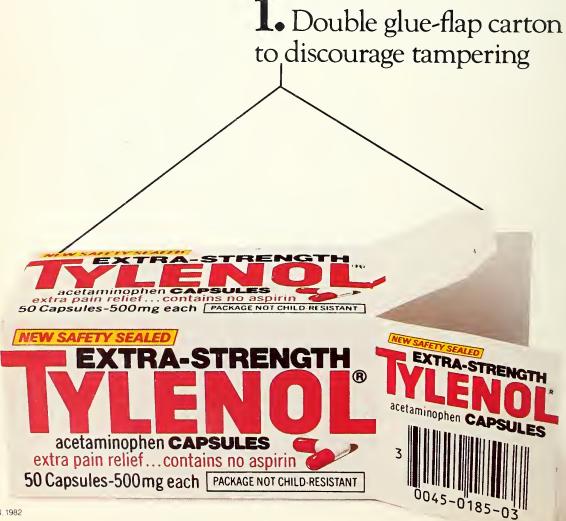
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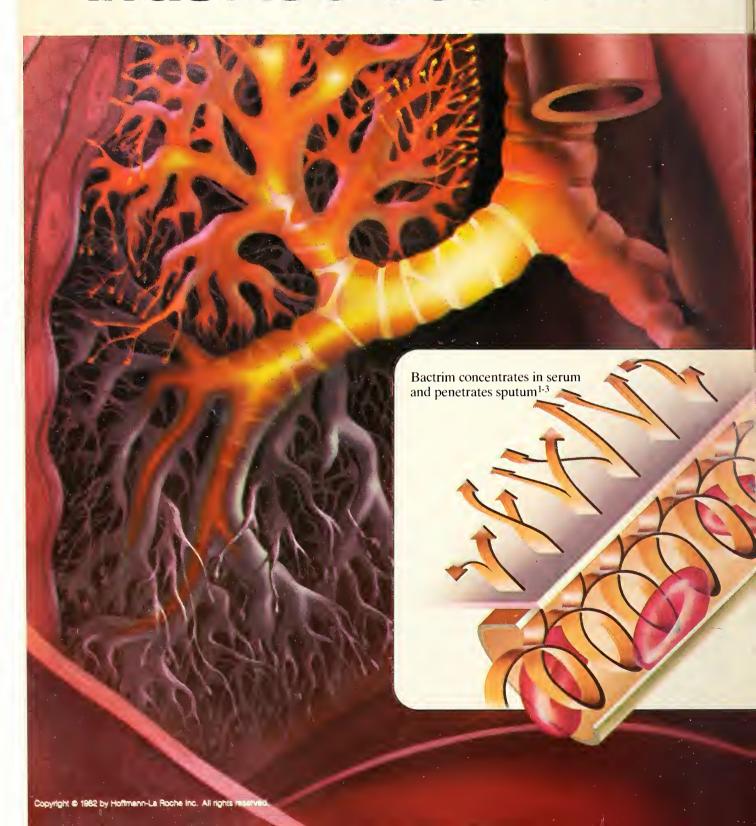


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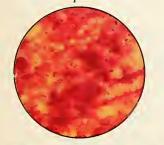
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Preceutions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

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S Clinicopathological Conference

Eleven Year Old Caucasian Male with Fever and Cervical Lymphadenopathy

Rande K. Short, M.D.*
Discusser

John F. Barlow, M.D.** Editor

Case #925 922

This 11 year old caucasian male entered Sioux Valley Hospital with a chief complaint of fever and swollen neck glands of three days duration.

This patient was well until 4 days prior to admission when he complained of fatigue and malaise. One day later he noted "swollen glands" in the neck which were very painful and tender. There was no history of sore throat or upper respiratory symptoms. He consulted a local physician who noted a low grade fever for which he prescribed aspirin and/or acetaminophen and erythromycin for a mild pharyngitis. The patient continued to feel poorly and one day prior to admission awoke with severe pain and tenderness in the swollen cervical lymph nodes, accompanied by a temperature of 104°F. The patient could swallow fluids without difficulty but did not want to eat solid foods. There were no other complaints. Before transfer, throat cultures and blood cultures were obtained. The blood culture revealed no growth and the throat culture revealed no significant pathogens. A white count before transfer was 21,700/mm3 (21.7 \times 10⁹/L), with 22% bands neutrophils. Erythrocyte sedimentation rate was 120mm/hr. A monospot test was negative. A PPD skin test of intermediate strength (mantoux) was negative. The patient was hospitalized and given cephalothin 500 mgs. every 6 hours and acetaminophen for temperature. Because of continued fever, the patient was transferred.

The patient's past history revealed no serious illnesses or hospitalizations. He had had streptococcal sore throat twice in the past year. Social and family history were unremarkable. PHYSICAL EXAMINATION: Temperature—103.2°F, pulse 132/min. and regular; respirations 22/min. and regular; blood pressure 88 systolic and 42 diastolic, weight 76 pounds. The patient was acutely ill. The neck was swollen associated with posterior auricular bilateral exquisitely tender lymph nodes with overlying erythema. Anterior cervical lymph nodes were moderately enlarged with moderate tenderness. There was no remarkable conjunctivitis on admission, although this developed later. The tonsils were enlarged and had exudate. The posterior pharynx was slightly

red. There was no tenderness over the parotids. There were a few inspiratory wheezes in the posterior right lung field. The heart was not enlarged. There was a sinus rhythm without murmurs. The examination of the abdomen revealed no tenderness, spasm, palpable organs or masses. The genitalia and extremities were unremarkable. Neurologic examination was within normal limits. LABORATORY DATA: Urinalysis - amber, clear, specific gravity 1.013, pH 6.0; protein 1+, negative for glucose, ketone bodies and hemoglobin; positive for bile; sediment 4-6 white cells/hpf. and a small amount of bacteria. Hemoglobin 11.9 gm/dl, hematocrit 36 vol/dl, normal red cell indices. Total leukocyte count 11,300/mm3 (11.3 × $10^9/\text{L}$) with 63% segmented neutrophils, 28% neutrophilic bands, 5% normal lymphocytes and 4% monocytes. The neutrophils showed toxic granulation and Dohle bodies. The red cells were normochromic, normocytic. The platelet count was 198,000/mm3 ($198 \times 10^9/\text{L}$). Erythrocyte sedimentation rate was 106 mm/hr. Lactic dehydrogenase (LDH) was 363 IU/ L (normal 0-270/L), aspartate aminotransferase (AST) was 110 IU/L (normal 0-60 IU/L), total bilirubin 9.1 mg/dl, with a direct of 6.9 mg/dl and indirect of 2.2 mg/dl. Total protein 4.9 gm/L (normal 5.5-8.3 gm/L), calcium 8.2 mg/dl (normal 8.3-10.3 mg/ dl). Cholesterol 183 mg/dl (normal 150-205 mg/dl). Alkaline phosphatase, inorganic phosphorus, glucose, blood urea nitrogen, creatinine kinase, creatinine, uric acid and gamma glutamyl transpeptidase (GGTT) were within normal limits. Electrolytes were as follows: sodium 131 meq/L, potassium 3.8 meq/L, chloride 104 meq/L, CO2 content 19 meq/L.

After admission the patient's therapy was changed to methicillin 200 mg/kg per day intravenously. The patient continued to spike irregular fever up to 103-105°F. A biopsy of a cervical lymph node revealed only chronic nonspecific inflammatory changes. A titer for salmonella group B was positive at 1:160 but did not rise in convalescence. Acute and convalescent titers for the following organisms were negative: Leptospira, Toxoplasma gondii, Herpes simplex, Group A beta hemolytic streptococcus (antistreptolysin 0), Cytomegalovirus, Epstein-Barr virus (both specific IgM and IgG viral capsid antigen and monospot tests), Brucella and Francisella tularensis. Test for HBsAG was negative. Multiple blood cultures were negative; wound cultures from the neck after surgery for the lymph node biopsy were negative. A fluorescent antinuclear antibody test (FANA) was negative. Multiple sputum cultures for mycobacteria and fungi were negative. Stool cultures for Salmonella, Shigella, Campylobacter jejuni, and Yersinia enterocolitica were negative. Viral cultures of throat and stool were

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negative. Immunoglobulins (IgM, IgG, and IgA) were within normal limits. Total complement, C3, C4 levels were within normal limits. Haptoglobin was within normal limits. Immunodiffusion titers for Histoplasma capsulatum, Candida, Blastomyces dermatitidis, and Coccididoes immitis were negative.

The patient developed marked erythema with a raw hamburger appearance of the throat. This was treated with tobramycin. The patient developed abnormal liver function tests with jaundice. The peak bilirubin was 10 mg/dl. LDH peaked at 645 IU/L, alkaline phosphatase rose slowly to 323 IU/L (normal 0-115 IU/L) and AST to 621 IU/L. The patient developed hydrops of the gall-bladder during the period of the abnormal liver function tests. These showed progressive descent toward normal over hospitalization and clinical jaundice disappeared. The spiking fevers ceased after nine days.

Chest film showed a large pleural effusion at the right base and some cardiomegaly. An upper gastrointestinal series was negative. A computer tomogram of the head was negative. Echocardiogram revealed a small pericardial effusion and a possible left coronary artery aneurysm. Electrocardiograms were within normal limits for age.

The patient did have an episode when he was unable to tolerate feedings and was only mildly responsive, very uncomfortable, and acutely ill before his temperature decreased. During this time he was somewhat somnolent but was able to answer questions. He was treated with aspirin therapy which was monitored with blood levels. The dose had to be increased considerably to maintain adequate levels. He was treated with hyperalimentation for several days because of vomiting and inability to tolerate feedings. He also suffered what was termed an oculogyric crisis and became opisthotonic and cyanotic. He was treated with diazepam and moved to the intensive care unit where he was given oxygen by mask. The patient gradually was able to tolerate feedings and had no recurrence of the seizures. The hemoglobin dropped to a low of 7.8 gm/dl during the course but the hemoglobin was 9.7 gm/dl by discharge. The white count and erythrocyte sedimentation rate declined to within normal range but reticulocyte count was 19.7% at discharge. This was not felt to be due to hemolysis, but to be secondary to regeneration of red cells by the bone marrow. The patient developed marked reddening of all of his mucous membranes, including the nose, lips, and throat and had some chronic mild epistaxis during his hospitalization. Toward the end of the hospitalization there was peeling of the skin of the hands and feet.

DR. SHORT: To review the case, an 11 year old male initially complained of fatigue and cervical adenopathy. He was initially treated for the above plus a low grade fever and pharyngitis with erythomycin and antipyretics. During his hospitalization, he displayed multi-organ disease involving integument, hepatic, hematopoietic, urinary, central nervous, lymphatic and cardiopulmonary systems. He remained febrile despite adequate antibiotic therapy. A thorough search for an infectious etiology was unsuccessful. Past medical history was significant for only two streptococcal sore throats in the past year and no history of any antecedent respiratory symptoms. Laboratory analysis revealed leukocytosis, elevated erythrocyte sedimentation rate (ESR), proteinuria, mild anemia, elevated liver enzymes, elevated bilirubin and hypoproteinemia. The patient developed a pleural effusion, hydrops of the gallbladder and possible left coronary artery aneurysm during his hospitalization. He also displayed central nervous system involvement with an oculogyric crisis and seizure activity. Towards the end of his hospitalization, he exhibited desquamation

of his hands and feet.

The initial differential diagnosis needs to include disease entities which could cause diffuse changes of the skin and mucous membranes, conjunctivitis, fever, cervical adenopathy, fatigue and malaise. The following three groups need to be considered:

I. INFECTIOUS

- A. Rubella
- B. Rubeola
- C. Roseola Infantum
- D. Rheumatic Fever
- E. Toxic Shock Syndrome
- F. Streptococcal Scarlet Fever
- G. Staphylococcal "Scalded Skin" Syndrome
- H. Rat-bite fever
- I. Leptospirosis
- J. Toxoplasmosis
- K. Yersinia Enterocolitis Infection
- L. Enteroviral Illness
- M. Exanthematous Rickettsial Disease (i.e. Rocky Mountain Spotted Fever)
- N. Primary Epstein-Barr Viral Infection

II. COLLAGEN VASCULAR DISORDERS

- A. Juvenile Rheumatoid Arthritis
- B. Systemic Lupus Erythematosis
- C. Reiter's Syndrome
- D. Acute Scleroderma
- E. Infantile Polyarteritis Nodosa

III. OTHER ENTITIES

- A. Stevens' Johnson Syndrome (erythema multiforme exudativum)
- B. Kawasaki's Disease (mucocutaneous lymph node syndrome)
- C. Acrodynia
- D. Behcet's Disease
- E. Drug Eruption with Fever

The case presented today most likely represents Kawasaki Disease. In 1967 Kawasaki presented his first paper outlining the criteria for diagnosis of mucocutaneous lymph node syndrome. (Figure 1)

Five of six of the criteria must be met with fever as one of the criteria. Today's case had all of them with the exception of an erythematous rash. Also, the patient's hospital course was consistent with the progression of Kawasaki Disease (KD). Additional findings seen in KD are pyuria, arthralgia, arthritis, diarrhea, abdominal pain, aseptic meningitis, myocarditis, hepatitis, obstructive jaundice, hydrops of the gallbladder, aneurysms of the coronary arteries, meatitis, tympanitis, photophobia, pneumonia, convulsions, encephalopathy, pleural and pericardial effusions, congestive heart failure, myocardial ischemia and/or infarction. Mucocutaneous lymph node syndrome is strictly a clinical diagnosis and is a diagnosis of exclusion. There are no specific lab-

Kawasaki Syndrome Principal Diagnostic Criteria

Fever, for more than five days Conjunctival injection Changes in the mouth Erythema, fissuring, and crusting of lips Diffuse oropharyngeal erythema Strawberry tongue

Changes in the peripheral extremities Induration of hands and feet Erythema of palms and soles

Desquamation of tips of fingers and toes approximately two weeks from onset of illness

Transverse grooves across fingernails two to three months after onset of illness

Erythematous rash

Enlarged lymph node mass > 1.5 cm in diameter

Associated Manifestations

Pyuria Arthralgia, arthritis Diarrhea Abdominal pain Aseptic meningitis Carditis Hepatitis Obstructive jaundice Hydrops of gallbladder

Figure 1

oratory tests, although acute phase reactants may be elevated. In this case, the ESR was 120 mm/hr, but note the normal haptoglobin. It is important to treat initially for a possible infectious etiology since there are reported cases of children misdiagnosed as KD who expired secondary to a potentially treatable disease. In today's case, other causes were effectively ruled out and appropriate antibiotics were used. This case also illustrates more unusual complications of KD. Hydrops of the gallbladder has only recently been noted^{9,10,13}. It is also seen with leptospirosis, scarlet fever, and familial Mediteranean fever. Recent studies have shown this complication may be followed by serial ultrasound studies and surgical intervention is rarely necessary^{9,10,13}. The etiology of this phenomenon is unclear. This patient also exhibited evidence of obstructive jaundice; possibly due to stasis in the biliary system. The central nervous system involvement was evidenced by an oculgyric crisis and change in level of consciousness. This likely represented an aseptic meningitis, although results of a lumbar puncture are not available. Sector scan revealed a possible coronary artery aneurysm which may be seen in up to 30% of patients. I will discuss cardiac involvement later.

At this time, the epidemiology of KD is not well

understood. No causative factors have been identified, although there has been a weak connection with antecedent upper respiratory illness. No bacterial or viral etiologies have been found. HLA typing has not been helpful. There does seem to be a higher incidence in Japanese and their descendents. 50% of the victims are under age two, while 80% are under age four8. The male to female ratio is 1.5:1. Cases have been reported throughout the world, the majority in Japan^{1,5,7,8,11,14}. The case incidence in the United States is approximately 1-2/100,000 in children less than eight years of age. There have been no seasonal variation, geographic locale, eating habits or other factors associated consistently with the disease. Case mortality is approximately 2% due mainly to cardiac involvement.

The clinical course and treatment of KD can be divided into three phases: acute, subacute, and convalescent. Important cardiac considerations are outlined. (Figure 2)

Kawasaki Syndrome Pathologic Findings at Autopsy Stage I (≤10 days after onset)

Acute perivasculitis of coronary arteries Microvascular angiitis of coronary arteries and aorta Pancarditis with pericardial, myocardial, endocardial inflammation

Inflammation with AV conduction system

Stage II (12-28 days after onset) Acute panvasculitis of coronary arteries Coronary artery aneurysms Coronary obstruction and thrombosis Myocardial and endocardial inflammation less intense

Stage III (28-45 days after onset) Subacute inflammation in coronary arteries Coronary artery aneurysms Myocardial, endocardial inflammation greatly depressed

Stage IV (50 days after onset)

Scar formation, calcification in coronary arteries

Stenosis and recanalization of coronary vessel

Myocardial fibrosis without acute inflammation

Adapted from H. Fujiwara. Y. Hamashima: Pediatrics 61:100, 1978

Figure 2

I. ACUTE PHASE: Days 1-10

During this phase, the classic findings of fever, conjunctival injection, oral changes, swollen hands and feet, rash, or adenopathy may be present. Additional findings of central nervous system irritability, urethritis, abdominal pain,

hepatitis, and hydrops of the gallbladder may also be seen. The ESR is usually elevated; however, platelet count is usually normal during this phase. Cardiac involvement is manifested as tachycardia or possible gallop rhythm. General supportive care is the baseline for treatment. Aspirin therapy (80-100 mg/kg/day), monitored with blood levels of aspirin should be utilized. One might consider initial sector scan from day 7-12 to detect coronary artery disease.

II. SUBACUTE PHASE: Days 10-40

During this phase, the patient may exhibit desquamation of the hands and/or feet. The ESR may start to return to normal. During this phase, the patient may exhibit a thrombocytosis with a platelet count greater than 450,000/ mm³. Some authors suggest continuing aspirin at 10 mg/kg/day for the next 6-10 weeks. Cardiac complications during this phase include congestive heart failure, arrythmias, pericardial effusion and mitral insufficiency. It is at this time that the highest risk of death is present. Myocardial infarction may occur secondary to coronary thrombosis or rupture of a coronary artery aneurysm. Some authors advocate a sector scan during this phase to look for a coronary artery aneurysm on days 28-35. Positive sector scans may be an indication for cardiac catheterizations. Approximately 17% of cases have coronary artery aneurysms. However, ½ to ²/₃ of these regress on repeat cardiac catheterizations in one year.

III. CONVALESCENT PHASE: Days 40 onward The ESR should continue to decrease. At this time, transverse grooves may appear in the nails during this phase. Some children may still exhibit persistent angina.

Effective treatment for KD awaits the discovery of the etiology and pathogenesis. Most authors agree that prophylactic aspirin therapy may help reduce cardiac complications which are associated with the thrombocytosis and platelet aggregation^{4,8}. Kato demonstrated that steroids might acutally increase the incidence of coronary artery aneurysms⁴. Early diagnosis and good supportive care are essential in the management. Surgical and medical complications need to be managed as they arise⁹. Management of the cardiac complications and coronary artery aneurysms are likely the most important factor^{3,4,12}.

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The majority of the deaths occur in this area. Serial sector scans need to be performed and, if positive, cardiac catheterizations need to be considered. Some authors have reported successful coronary artery bypass graft in selected cases. Further clinical trials need to be performed to help outline further care of patients with KD.

The prognosis for KD is generally quite good. It is usually a self-limited disease with no residual deficits. However, currently there has been only a 15year follow up. Some authors have looked for an association of the vasculitis of KD with idiopathic adult vascular sclerosing diseases such as fibromuscular dysplasia of the renal artery. Takayasu's Disease, and primary pulmonary hypertension. So far, no definite association has been found⁶. Some authors feel that KD is an extension of infantile polyarteritis nodosa². There is good pathological evidence to support this. In one study by Melish, et al8, 85 children were followed for 2-9 years after recovery. Four of them described exercise-induced angina; two of which had residual aneurysms. Seven others had electrocardiographic changes and one was hypertensive. At this time, their course is unpre-

In conclusion, physician awareness and longitudinal follow up will be essential in the diagnosis and management of this disease.

Dr. Short's Diagnosis Kawasaki Disease

DR. BARLOW: This case was presented to increase awareness of this puzzling entity originally described in Japan. It is difficult to make the diagnosis of KD until many of the features become apparent. The treatment for the disease is aspirin since corticosteroids are contraindicated according to the Japanese. Aspirin therapy in KD is supposed to be an antiplatelet agent which may prevent thrombosis of the coronary arteries. However, in febrile illnesses of children many feel that aspirin is contraindicated because it may precipitate Reye's syndrome. What can one do about this dilemma?

*DR. THATCHER: Yes, national pediatric groups have published data along with federal agencies suggesting that aspirin not be used if a child has nonspecific, viral-like illness suggesting influenza or chicken pox. Dr. Gellis does not feel there is sufficient evidence that aspirin does indeed precipitate Reye's syndrome. Regardless, many pediatricians feel that aspirin is not helpful in viral illness and may be potentially toxic in some cases. Unless the fever is quite high, there should be no treatment. There is even some evidence that fever is beneficial in inhibiting growth of some microorganisms.

**DR. FRANK FOSS: How high is a high fever?
DR. THATCHER: That is a difficult question to

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answer and I guess I would have to say 106°C. DR. SHORT: What about treatment by sponge bath? DR. THATCHER: That can be done, but it is not very pleasant for the child. Also, it may be the rapid changes in temperature whether increase or decrease, which causes febrile convulsions and not the absolute degree of fever. I must admit that this is a difficult problem. Aspirin does relieve the discomforts associated with viral illness and parents are quite anxious with a child with a high fever. Being told that no treatment is necessary is a difficult concept for them to accept.

*DR. JERRY SIMMONS: The alternative therapy, acetamenophen, is also a toxic drug to the liver and the kidneys.

**DR. PAT PETERS: What is the cause of the anemia in this disease?

DR. THATCHER: It is probably secondary to suppression of bone marrow function. The thrombocytosis and rise in reticulocyte count in the latter stages of the disease, as in this case, often represents a regenerative phenomena.

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Future Meetings

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- Drug Therapy Symposium, Holiday Inn, Minneapolis, MN, Feb. 16-17, AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Cedar's Medical Center's Fifth Annual Oncology Update, Cedar's Seminar Ctr., Miami, FL, Feb. 18-19. Fee: \$165. 111/2 hrs. AMA Category I credits. Contact: Karen Fuson Buchsbaum, Dir. of Pub. Relations, 1400 NW 12th Ave., Miami, FL 33136. Phone: (305) 325-5511.
- ENT Problems for Primary Care Physicians, Sheraton-Ritz Hotel, Minneapolis, MN, Feb. 25-26. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

March

Clinical Therapeutics—1983, Radisson Plaza Hotel, St. Paul, MN, March 3-5. Fee: \$200. 21 hrs. AMA Category I credits. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

- Advanced Cardiac Life Support, Burlington, IA, April 1-3. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- IX Annual Conference on Perinatal Medicine, Des Moines, IA, April 5-6. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, 1A 52242.
- Ophthalomology Clinical Conference, U. of Iowa, Iowa City, IA, April 6. AMA Category I credits. Contact: Richard Caplan,

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- Behavioral Medicine, Coffman Mem. Union, U. of Minn., Minneapolis, MN, April 6-7. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Evaluation and Treatment of the Patient with Acute and Chronic Pulmonary Disability (Physical Therapy), U. of Iowa, Iowa City, IA, April 7-9. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.
- Annual Spring Meeting of the ASCP/CAP, Hyatt Regency Hotel and Radisson Hotel, Chicago, IL, April 9-14. Contact: Michael Kelleher, ASCP Customer Serv., 2100 W. Harrison St., Chicago, 1L 60612. Phone: (312) 738-1336.
- Otolaryngology Clinical Conference, U. of Iowa, Iowa City, IA, April 15. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, April 21. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Allergy and Clinical Immunology, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, April 21-23. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Current Management of Vitreo-Retinal Disease, Holiday Inn Downtown, Minneapolis, MN, April 25-26. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.



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Clinicopathological Conference Seventeen Year Old Caucasian Male with Recurrent Abdominal Pain

S Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Saturday, November 20, 1982, at the Howard Johnson Motor Lodge, Sioux Falls, South Dakota. Following are the items of business transacted at this meeting.

- 1. LEGISLATION. The Council voted to support legislation amending the Coroner's statute; to introduce legislation which would allow a physician to treat a minor without prior consent of a parent when in the physician's judgment an attempt to secure consent would increase the risk to the minor's life or health; to support legislation to strengthen the DWI statutes; and to support the concept of child mandatory restraint systems if such legislation is introduced.
- 2. POLITICAL AWARENESS. The Council directed the executive office to contact all member physicians through the district medical societies and designate a "key contact person" for each legislator in the state.
- 3. MEMBER SURVEY. The Council directed the executive office to survey the membership regarding the needs and concerns of the members. This will be done in late February or early March.
- 4. HONORARY MEMBERSHIP. Dr. E. A. Pasek, Sioux Falls, was voted honorary life membership in the State Medical Association.
- 5. VISA-MASTERCARD BANK PLAN. The Council directed the executive office to proceed to implement the Visa-MasterCard Bank Plan for member physician offices in South Dakota. This plan will allow doctors' offices to utilize these credit cards in their offices for payment of bills by patients at a lower service charge than individual offices are charged.
- 6. LEVELS OF R.N. DEGREES. The Council reiterated its stand supporting the three levels of nursing.

Family Physician

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S Clinicopathological Conference

Six Week Old Infant with a Dry Hacking Cough

Rick L. Plummer, M.D.* Jerome M. Blake, M.D.** Discussers

John F. Barlow, M.D.*** Editor

Case #155 2387

This six-week-old infant was referred to McKennan Hospital with a history of chronic cough. He became ill three weeks prior to admission at which time he apparently began to have cramps and developed a dry hacking cough. Initially, he was treated with a formula change from a cow's milk formula to a soybean base formula without any change in the cough. The cramps apparently disappeared. His cough increased in severity and became episodic occurring in frequent episodes every 20 minutes to an hour. He had no history of a fever, apnea, or perioral cyanosis with the coughing. There was no history of conjunctivitis at the onset of his illness. One week prior to admission a chest x-ray revealed a very mild perihilar infiltrate. His blood count was normal. He was treated symptomatically without antibiotics.

His past medical history revealed that he was an apparent normal full term male infant delivered spontaneously by the vaginal route. There was no accurate history because he was adopted at one week of age.

Physical examination on admission: temperature 98.4°F. Pulse 144/min., respirations varied between 35 and 40/min., weight 3.3 kg. and height 41.2 cm.

He was an alert, active, thriving six-week-old child who was quite tachypneic and had subcostal, sternal, and intercostal retractions. Examination of the skin was normal. Examination of the head and neck revealed a normocephalic head, a soft anterior fontanelle, normal appearing tympanic membranes and no evidence of conjunctivitis. His nose, mouth and throat were all normal. The neck was supple. There was no cervical lymphadenopathy, masses or thyromegaly. There were moist rales heard throughout the chest bilaterally. The most remarkable finding was the child's coughing episodes which were very moist and staccato in nature. The coughing episodes continued for some period of time. He did not turn cyanotic nor did he whoop with the cough. The heart

was not enlarged and there were no cardiac murmurs. Examination of the abdomen and extremities and neurologic examination were within normal limits.

His initial laboratory studies revealed a hemoglobin of 10.5 gm/dl, hematocrit of 31 vol/dl with normal red cell indices. White count was 30,700/mm3 ($30.7\times10^9/\text{L}$) with 14% segmented neutrophils, 1% neutrophilic bands, 37% lymphocytes, 15% monocytes and 30% eosinophils. A chest x-ray was obtained which revealed a diffuse alveolar and interstitial type pneumonia radiating from the perihilum into both lungs extensively.

HOSPITAL COURSE: He was admitted to the Intensive Care Unit and initially placed on supplemental oxygen. Appropriate cultures were obtained, and he was placed on an antibiotic and over the course of the next week rather rapidly improved. A sputum culture grew normal flora and a small amount of Escherichia coli and Enterobacter cloacae which were thought to be colonizing organisms. An intermediate tuberculin test PPD was negative. A cold agglutinin titer was negative. A sweat chloride was normal. Total protein was 8.1 gm/L with an albumin of 4.0 gm/L and a globulin 2.5 gm/L. Acute and convalescent titers for influenza A and B, parainfluenza viruses, adenoviruses. Respiratory syncytial virus.

Herpes simplex virus, Chlamydia psittaci and Mycoplasma pneumoniae were negative. Sputum culture for Chlamydia trachomatis was positive and an acute serum antibody titer to chlamydia by microimmunofluorescence was positive at 1:4906.

DR. PLUMMER: I would like to begin the discussion today with a general overview of a neonatal pneumonia. A simple, but useful schema for discussion divides neonatal pneumonia into 3 classes which are determined by the route of acquisition and by the age at which the infant presents. These 3 categories are transplacental pneumonitis, aspiration pneumonitis, and acquired pneumonitis.

Transplacental pneumonias are those types which are acquired in utero and which are present in the first few hours of life. These pneumonias may be part of the generalized congenital infections such as

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with Cytomegalovirus, Herpes simplex virus, Rubella virus, Toxoplasma gondii, or Listeria monocytogenes. Mycoplasma and syphilis have also, caused fatal pneumonias in this setting. Often the pneumonia is overshadowed by more ominous findings such as neurologic abnormalities hepatosplenomegaly, rashes, petechiae or other teratogenic effects.

The next class is that of aspiration pneumonitis which is acquired in the immediate perinatal period and which presents itself in the first few days of life. This is felt to evolve from an aspiration of material from the cervix or from aspiration of amniotic fluid before and/or during delivery. Another situation which should be included is that instance in which the fetus aspirates maternal fecal contents. With the exception of this last, it is felt that most infants with aspiration pneumonitis have not swallowed infectious material and no antibiotics are indicated. This does include the condition of meconium aspiration. The pneumonia which develops is on a chemical basis. When there are bacteria causing this early onset pneumonia, the most common pathogens seen are the Group B beta hemolytic streptococcus, Group D streptococcus, pneumococcus or coliform organism. These infants may develop severe respiratory distress and shock in the first 12 hours of life.

The third category encompasses the acquired pneumonias and is delayed in onset. These are those pneumonias which are contracted during the postpartum period and usually after the first week of life. These pneumonias may be viral, bacterial, or chlamydial in etiology. There are most frequently bronchopneumonic or interstitial in pattern. Some authors feel that the respiratory syncytial virus is the most important pathogen in this category. The parainfluenza and adenoviruses may be prominent causes of bronchiolitis and pneumonia. Other agents known to be involved are echovirus type 22 and influenza viruses types A and B. The most common bacterial pathogens seen in this group are Staphylococcus aureus and the coliform organisms. Complications such as meningitis, osteomyelitis, and septicemia are often seen. Chlaymdia trachomatis pneumonitis fits loosely into this category and will be discussed extensively below.

The clinical signs of pneumonia in the neonate are often subtle. The earliest signs may be listlessness, report that the child is not feeding well, poor color, tachypnea, dyspnea, cough or irregular breathing. A decreased temperature may be noted. Increasing respiratory distress, flaring of the alae nasi and retractions may be seen. Actual dullness to percussion may be difficult to demonstrate; however when present, it does indicate consolidation.

Decreased breath sounds may be heard along with rales and wheezes. A brassy cough may indicate a viral infection.

General diagnostic measures which should be employed in the differential diagnosis of pneumonia include a chest x-ray, white blood cell count and culture of blood, trachea, ear canal, nasopharynx and throat. A lung puncture should be considered in infants who have definite consolidation and who do not respond as expected to therapy. Material obtained from this should be gram stained and cultures obtained for multiple microorganisms. The laboratory should be consulted as how the material should be handled.

It is often extremely difficult to determine the etiology of the pneumonia from the x-ray pattern. Pneumonia due to Staphylococcus aureus often demonstrates a consolidating bronchopneumonia with pneumatoceles and empyema. Klebsiella pneumonia presents a picture of a lobar infiltrate associated with a bulging fissure. Listeriosis, in its classic form, gives a miliary type bronchopneumonia. Pneumocystis carinii gives a bilateral ground glass haziness which extends outward from the hilar regions. This entity, in the newborn, is rare in this country but has been seen in epidemic form in Europe. It is seen in the USA in patients who are immunosuppressed as well as male homosexuals and hemophiliacs. The picture as seen with chlamydial pneumonia will be discussed below.

In general, with pneumonia in the first month of life, a bronchopneumonic type infiltrate is most commonly seen. A leukopenia with increased band forms may often be seen in early onset bacterial pneumonia.

I would like now to focus more specifically on the organism Chlamydia trachomatis (CT). This organism takes its name from the Greek work "chlamys" which means a cloak draped from the shoulder. This describes the draping of the inclusions around the host cell nucleus. CT was first demonstrated by Halberstadter and Von Prowazek in 1907 as intracytoplasmic inclusions in the conjunctival scrapings from a patient with trachoma. This organism was not grown in cell cultures until 1965. It was not associated with pneumonitis until a case was described by Schachter in November of 1975 when a child, both of whose parents had proven genital tract infections with CT, developed pneumonitis following inclusion blennorrhea (inclusion conjunctivitis of the newborn infant). CT is felt to be one of the most prevalent sexually transmitted diseases. It accounts for up to 40-50% of non-gonococcal urethritis in men and may account for up to 20% of nongonococcal pelvic inflammatory disease in females. Many females are felt to be asymptomatic carriers.

The organism is probably transmitted to the infant during delivery, giving both the opthalamic and respiratory complications.

CT belongs to the family Chlamydiaceae with one genus Chlamydia in which are two species C. trachomatis (CT) and C. psittaci. The major reservior for CT is man and the organism is pathogenic for columnar epithelial cells. The organism is an obligate intracellular parasite but is not a virus. In fact, some consider it a bacterium. The reasoning for this is based on the findings that the organism has both RNA and DNA, possesses a cell wall, and divides by binary fission. It is susceptible to a number of antibiotics including sulfonamides, erythromycin, and tetracycline. It has been likened to an "energy parasite" as it has no mitochondria or ATP. CT has a complicated life cycle with an intracellular and extracellular phase. Several serotypes have been identified. A through C have been associated with trachoma, and D through K with inclusion conjunctivitis and pneumonia in infants, whereas L-1,2,3 have been associated with lymphogranuloma venereum.

The organism does form cytoplasmic inclusions that displace the host cell nucleus. The inclusions contains glycogen like material which stains with iodine, Giemsa and fluorescent antibody stains. It is the staining of the glycogenlike substance in the inclusions with iodine that differentiates CT from C. psittaci.

Transmission of CT in adults is felt to be horizontal and primarily through the sexual route. Transmission of the organism in infants is felt to be vertical from mother to infant during delivery. Transplacental, postnatal, and horizontal transmission from infant to infant have not been demonstrated. The most important risk factor for the infant is contracting the organism from an active cervical infection in the mother prior to delivery. The prevalence of cervical infection in women has been estimated at from 2 to 21%. The infection is known to be most prevalent in women who are young, unmarried, primiparous and black. Chlamydial infection, however, is found in 4.8% of non-pregnant, female, college students. The transmission rate of mother to infant ranges from 54-70% when one includes nasopharyngeal infections, pneumonitis, and conjunctivitis. The risk of obtaining conjunctivitis from the infected mother is approximately 35%. The risk of contracting pneumonia is approximately 15%. Approximately 20% of infants will obtain an asymptomatic nasopharyngeal infection and approximately 20% may also have a positive rectal culture which at this time has not been proven to be of any significance. The nasopharynx does appear to be the most consistent site from which to isolate CT. Studies have shown that pneumonia does not necessarily follow conjuctivitis or nasopharyngeal infection, but up to 33% of infants with nasopharyngeal infection may develop pneumonia. CT is the most common cause of neonatal conjunctivitis, and, hence the use of an appropriate eye ointment such as erythromycin or tetracycline is preferable for neonatal ocular prophylaxis since silver nitrate does not prevent the development of chlamydial conjunctivitis.

CT pneumonia has been intensively investigated in the mid and late 1970's. This syndrome is not new and, as early as 1941, Botsztejn described a syndrome which he called "pertussoid eosinophilic pneumonia of infancy." The salient features of pneumonia due to CT are as follows: the age of onset is somewhere between 2-12 weeks. It is characterized by absence of fever, gradual onset, nasal congestion, staccato cough, poor weight gain, and sometimes an inclusion conjunctivitis. Physical findings reveal tachypnea and rales. Wheezing is felt to be uncommon but may occur. Because of this, CT pneumonia probably should be included in the differential diagnosis of bronchiolitis and bronchial asthma. Examination of the chest film reveals hyperinflation and variable alveolar and interstitial infiltrates. Laboratory data reveals an eosinophilia greater than or equal to 300/mm3; an elevated IgG greater than or equal to 500 mg/dl, and an IgM of greater than or equal to 110 mg/dl. Chlamydial culture of the nasopharynx should be positive and one should find a chlamydial serum antibody titer greater than or equal to 1:32 as measured by the microimmunofluorescent test.

Viruses often may be isolated concomitantly. Cytomegalovirus is one of the most frequent isolates reported in the literature but respiratory syncytial virus, enteroviruses, adenoviruses, and rhinoviruses have all been isolated. However, in all of these instances the evidence is indicated that the over-riding picture is that of pneumonitis secondary to CT. Most infants recover uneventfully, however, scarring and compromised pulmonary function may develop.

Lung biopsy of infants with CT pneumonia reveals an interstitial infiltrate of lymphocytes, plasma cells and eosinophils with fibrous tissue proliferation, alveolar wall and peribronchial thickening. A mouse model has also been developed to aid in the study of this disease.

The Giemsa stain of epithelial cells obtained by scraping may still be helpful in the diagnosis of eye infections. However, the accuracy ranges from 30-95%. As the organism is an obligate intracellular parasite, it must be grown in the cell culture. Nasopharyngeal secretions are obtained in the same manner as for pertussis with the wire alginate swab

inserted through the nares. Specimens should be cultured immediately if possible and if they cannot be sent within 24 hours, they should be maintained on dry ice. The microimmunofluorescent test for antibodies in the serum is very sensitive and type specific. A paired serum should be obtained whenever possible. One may also test for chlamydial IgM antibody in tears.

Treatment has been shown to shorten the duration of the illness and to halt shedding of the organism. One may use erythromycin ethylsuccinate or estolate at 30-50 mg./kg. per day in 4 divided doses for 2-3 weeks. Alternatively, one may use sulfasoxizole, 150 mg./kg. per day. The use of trimethoprim/sulfame thoxazole is under study; however, it appears to be promising for CT. Approximately 80% of patients are felt to have good response to therapy with these agents.

Dr. Plummer's Diagnosis: Neonatal Pneumonia due to Chlamydia Trachomatis.

DR. BLAKE: The chest films are very remarkable. The hyperinflation, typical of this disease is hard to see but the perihilar infiltrate and patchy infiltrate through the rest of the lung fields is very striking. (Fig. 1)

*DR. B.W. LARSON: It said in the protocol that you obtained a sputum specimen. How did you do this?

DR. BLAKE: This was an induced sputum obtained by catheter suction by the respiratory therapy department.

**DR. B.T. PITT-HART: Do patients with urethritis develop antibody, and in this case, was the antibody IgG and IgM?

DR. BARLOW: Patients with urethritis may or may not develop antibodies and determining a rise in titer is difficult. A specific test for local or systemic IgM antibody would be helpful in diagnosis were it available. In neonatal pneumonia, a characteristic immune response occurs. Your point is also well taken that the high titer of antibody in this case could still represent maternal IgG antibody which can cross the placenta but this is quite a high titer at six weeks of age. If we knew that the IgM antibody was present, it would be more diagnostic since IgM



Figure 1
Chest film showing diffuse patchy infiltrate with hilar accentuation

antibody does not cross the placenta whereas IgG antibody does.

DR. BLAKE: There was a syndrome of infant pneumonia with eosinophilia presumed to be due to milk allergy because the presence of milk precipitins were demonstrated in the infant serum. I wonder whether some of these cases were not Ct pneumonias.

***DR. L.G. THATCHER: I believe one difference would be that in CT. pneumonia the patients are not nearly as sick and do not show the failure to thrive which is seen in the patients with allergic pneumonia caused by milk.

DR. BLAKE: As Dr. Plummer has nicely stated, neonatal pneumonias and infections have been broken down into those which are transmitted transplacentally, those acquired in the birth canal and those acquired after birth from personnel as nosocomial infections. Nurses have been blamed for transmitting many infections to infants. The breakdown of infections has been into so-called early onset and late onset. The early onset infections have been caused by such organisms as Group B streptococci, Listeria monocytogenes, coliform organisms (such as Escherichia coli), Neisseria gonorrhoeae, and Herpes simplex. These early onset infections usually are manifested within the first week after birth. They are presumed to have been contracted during the birth process or rarely transplacentally. Supposedly the late onset infections are nosocomial and are caused by similar organisms. However, is this true? Here we have a disease that is in the late onset category discussed today. This is CT pneumonia, yet this organism is acquired during the birth process from the cervix. Hepatitis B also occurs in the infant as a late onset disease but is probably acquired around the time of delivery. There is even a rec-

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ommendation to give hepatitis gamma globulin and possibly hepatitis vaccine to these infants of mothers who possess HB_sAg. In other words, I am questioning whether we know the exact epidemiology of neonatal infections and whether we can classify them as nicely as we have in the past.

DR. BARLOW: Because of the excellent discussion that has already occurred. I will just brush over a few major points. We will be able to culture Chlamydia trachomatis routinely in the near future. At the present time we have been working in the viral laboratory trying to perfect our techniques. We grow the organism on McCoy cells and detect the presence of growth by an iodine stain for the glycogen vacuole which occurs in the cytoplasm of the cells as described by Dr. Plummer. A suitable site for culture in the infant is the nasopharynx when one is looking for CT. Lung puncture may be required. If the patient develops inclusion conjunctivitis a scrape of the lower eyelid for Giemsa stain and culture is required. A physician used to dealing with the eye should obtain this specimen. In adults with sexually transmitted disease the urethral or cervical secretions are adequate for culture. The swabs should be calcium alginate swabs on metal wire because wood is inhibitory to the growth of chlamydia. Special transport media using sucrose phosphate or tryptose phosphate with gentamicin will be necessary. The ordinary viral transport media are not satisfactory. The specimen can be refrigerated for up to 24 hours but must be frozen -80° C to be preserved after that time.

As Dr. Plummer suggested, chlamydia are obligate intracellular parasites. Because they possess RNA and DNA and have a bacterial-like cell wall, they are not viruses. A very important clinical property is their susceptibility to antimicrobial agents such as tetracycline, erythromycin and sulfonamides. The organisms have a life cycle of 48 hours. The infectious particle is a metabolically inactive elementary body (350 nm) which attaches to heat sensitive receptors on the cell and is phagocytized. Within the cell, reticulate or initial bodies (800 nm) are produced. These are metabolically active and divide by binary fission. As maturation occurs more elementary bodies are produced and the cell rupture occurs releasing elementary bodies to attack other cells.

One of the two genera of the chlamydial organisms is Chlamydia psittaci which is a pathogen for birds and many other animals. It can cause a severe penumonia in humans which can be fatal. It is treatable with antibiotics.

As has been suggested, Chlamydia trachomatis has many serotytes. L1, 2, 3 cause lymphogranuloma venereum. In the late stages this disease can produce

rectal stricture and severe edema of the genitalia. It is relatively rare in this country. A characteristic histologic picture in the inguinal lymph nodes accompanies this disease and aids in diagnosis. Blinding hyperendemic trachoma is caused by strains A, B, Ba, C. This is a great problem in many parts of the world but not common in this country. However, the strains of CT, D-K, cause a great many infections. These include inclusion conjunctivitis in adults and neonates, the pneumonia discussed today in newborns, as well as other neonatal infections such as an otitis media and myocarditis. This organism is responsible for many cases of nongonococcal urethritis and postgonococcal urethritis in adults. It is a very common cause of salpingitis and the pelvic inflammatory disease syndrome. It has been estimated that pelvic inflammatory disease from this organism is more common than any of the other agents reputed to cause this condition. It may even cause the perihepatitis of Fitz-Hugh and Curtis which had previously been thought to be only caused by gonococcus. A recent study suggested that premature labor and perinatal fetal death can be related to cervical infection with CT. If this is confirmed, the organism becomes a terribly important one to study further.

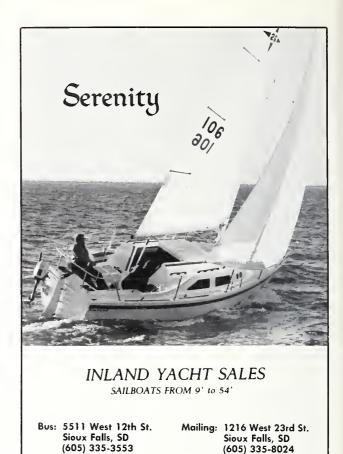
At the present time probably the best method of diagnosis is isolation on tissue culture which requires 48-72 hours. The direct Giemsa stain from tissue is usually only helpful in inclusion conjunctivitis of the newborn and is not very helpful in genitally acquired infections. Other tests using the detection of locally produced IgM may make diagnosis easier in the future. However, culture is now the method of choice.

FINAL ANATOMIC DIAGNOSIS: NEONATAL PNEUMONIA DUE TO CHLAMYDIA TRACHOMATIS.

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Chief, Ambulatory Care Services

Opening anticipated February, 1983, for Chief, Ambulatory Care Services at Sioux Falls, SD, VA Medical Center (GM&S 248 bed hospital). Prefer applicant who is board eligible or certified in internal medicine or family practice. Sioux Falls VAMC is a major teaching affiliate of the University of South Dakota School of Medicine. Academic appointment available to qualified applicants.

Contact: Chief, Medical Service
Veterans Administration
Medical & Regional Office Center
P. O. Box 5046
Sioux Falls, SD 57117

SFeature D

Becoming a Doctor

A Reaction by John M. Rud, MS II USD School of Medicine 1982

I believe that for the eternity of my life I shall always recall a certain conversation I had with a fellow freshman student. He was in the midst of one of his great tirades where complaints spewed forth as water from a lawn sprinkler. Quietly I would sit through these episodes as he flitted from "boring, irrelevant course work" to "the lousiest instructor in all of medical schools." This time, noticing my silence again, he asked of me, "What really burns you up about school, John?" "To be perfectly honest," I replied, "I am all too thankful to be here to find anything to complain about." "You're crazy," he raved. "It's not normal not to complain."

Entering the profession of medicine has required each of us to make alterations to our lifestyles. While these changes cause much distress and lament among medical students nationwide, I sometimes wish that they could find the rewards of these adjustments.

Where else could they find the friendship of 63 other students so inviting? Where else can you discover an ingenuity you never thought you possessed? Where else can one learn to find pleasure in one's own company? And most of all, where else can one obtain access to the knowledge and art of this privileged life called medicine?

The anatomy lab or any other of the processes involved in our transition to medicine has not caused me to lose my feelings and emotions. To lose the gifts of empathy and kindness during this four years would be a fate worse than death. No one—no instructor, no course, no author—is going to take them away from me. For to take those would be to kill the child within me and this world should never be looked at unless it is through the wonderfully curious eyes of a child. Perhaps, as my classmate said, I am crazy, but maybe he has already lost his "child" while what lies around me is still a joy.

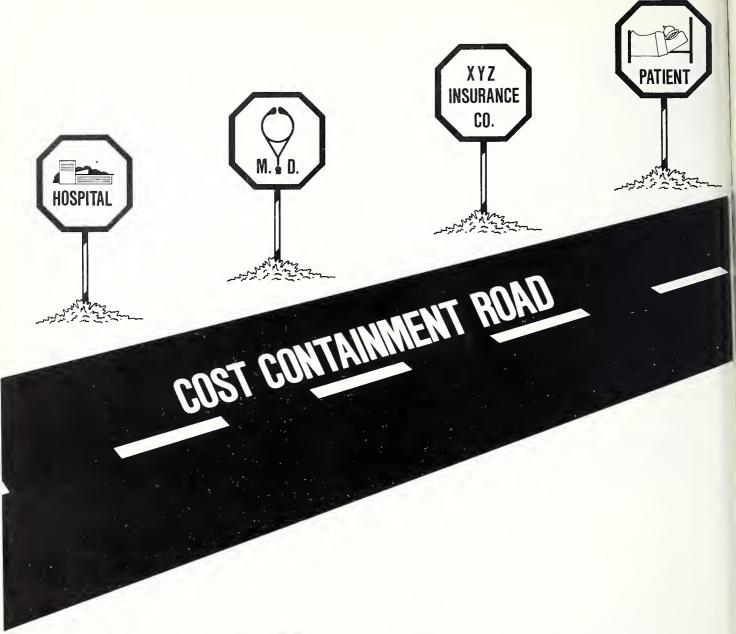
This reaction paper was written as a part of a course entitled Basic Interviewing Skills. Students were required to give their reaction to several chapters of a book entitled "Patient Interviewing: The Human Dimension" by David E. Reiser, M.D. and Andrea Klein Schroder, M.S.W. This reaction paper was written about Becoming a Doctor.

South Dakota Society Of Pathologists

Officers for 1982-83

Jerry L. Simmons, M.D., President Thomas E. Henry, M.D., Vice President Beth L. Johnson, M.D., Secretary-Treasurer





Who gets hurt if health insurance is priced out of the market?

A question that all of us must consider!

Health care cost containment is everyone's responsibility

SOUTH DAKOTA BLUE SHIELD

1601 West Madison Street Sioux Falls, South Dakota 57104



S President's Page D



The 102nd annual meeting of the South Dakota State Medical Association will be in Sioux Falls. The date is June 2-5, 1983.

Our Commission on Professional Liability, chaired by Morris Radack, M.D., has suggested that we present a Risk Management Seminar. The speakers are excellent. They are top people in their fields.

If you attend the entire session on Friday afternoon and Saturday morning, St. Paul Fire and Marine Insurance Company will give you a 10% reduction in your malpractice insurance premium

for one year, if they carry your insurance.

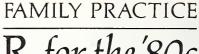
We expect our largest annual meeting attendance ever. Many physicians have already indicated that they plan to attend. Preference is given to our own members, of course, but please let our executive office know of your plans to attend as soon as possible.

I encourage you to take advantage of this chance to reduce your expenses, attend our annual meeting, obtain CME credit and perhaps learn to deal better with the frustrating legal aspects of medical practice.

Durword M. Laugmo

Durward M. Lang, M.D., President South Dakota State Medical Association

S Chapter News



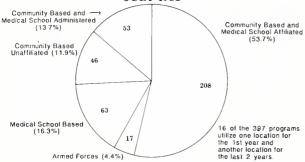
R for the 80s





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Fails, SD 57105

387 ACCREDITED FAMILY PRACTICE RESIDENCIES JULY 1982



The American Academy of Family Physicians

Results of Annual Survey of Family Practice Residency Programs

July, 1982

I.	Programs:	
	A. Total Accredited Programs:	387
	 Newly accredited programs that have 	
	not yet accepted residents	2
	B. Program Structure Types	
	1. Community Hospital Based 46	
	2. Community Based & Medical	
	School Affiliated 208	
	3. Community Based & Medical	
	School Administered 53	
	4. Medical School Based 63	
	5. Military Program 17	
11.	Residents:	
	A. Total Residents	7,204
	1. Total First Year Residents 2,578	, ,
	2. Total Second Year Residents 2,413	
	3. Total Third Year Residents 2,213	
	B. Total Approved First Year Positions C. First Year Fill Rate	2,657 97%
	D. Increase/Decrease Class Size by Year	
	1980-81 1981-82 1982-83	3
	Class of '83 $2,365$ $2,302$ $2,213$	_
	Class of '84 $$ 2,489 2,413	
	Class of '85 $$ 2,578	

A. Total July, 1982 Residency Graduates

residency programs since January 1, 1970

B. Total graduates from family practice

Huffington Library

An information nerve center for America's family doctors was dedicated in Kansas City, August 14, 1982. The new unit is the Herb L. Huffington Memorial Library, an information resource center that will enable family physicians across the nation to call on a toll-free telephone line and request information on any professional matter from the latest information on diagnosis or therapy to research in progress. It also will be a national repository for historical data regarding the medical specialty of family practice, including a collection of audio and videotaped interviews with early leaders of the specialty.

The library will be under direction of Patricia Butler, Ph.D., a professional medical librarian. The ultimate goal will be to:

- Develop a collection of current journals, books, and reference materials on family practice. Prepare and distribute bibliographies for immediate use by family physicians in disease management and patient education;
- Extend the basic reference services beyond the library site by doing data base searches via the MEDLINE and AVLINE computer systems; and
- Inform family physicians of research in progress, before it reaches print, and coordinate creation and maintenance of a computer data base of ongoing research projects in family practice.

"We already have one eye on the future, and expect soon to go into remote search services, to prepare and distribute bibliographies and abstracts from the literature for the use of family doctors, and develop other services to keep family physicians current with the knowledge explosion," Dr. Butler said.

"What we have to offer is the first-ever library facility expressly for the nation's family physicians, to help them in their practices to better serve their patients. We don't expect to duplicate the services of local medical libraries, but we want to tailor for the family physician the information that he or she needs specifically—and do it quickly, efficiently, courteously and inexpensively."

Dr. Butler does not expect to duplicate the services of local medical libraries, but plans to offer family practitioners information tailored to their needs quickly and inexpensively. In addition to standard reference works on family practice plans MEDLINE and AVLINE, the library staff hopes to prepare bibliographies for patient education. Call 1-800-821-2512—Librarian Butler awaits.

Member New Chamber President

2,183

12,834

SDAFP Past-President R. W. Friess, M.D. was installed as president of the Sioux Falls Area Chamber of Commerce in November. He had been elected to the President-Elect post the previous month, and moved shortly thereafter into the presidential chair due to a transfer vacancy. We wish him well!

III. Residency Graduates:

S Medicine

Hyperactivity—By Any Other Name

Roy C. Knowles, M.D.*

ABSTRACT

Through the use of clinical examples an effort is made to remove some of the confusion which appears to surround this disorder so that it may be recognized as a satisfactorily treatable condition.

It has been said, though the author has not looked up the reference, that some researcher over fifteen years ago found that the literature listed fifty-four different titles or names for the condition which we most often call hyperactivity or hyperkinesis or attention deficit disorder. These names appear to develop out of the particular interest or orientation of the examiner or author. The emphasis thus falls on such things as various kinds of learning disabilities, various kinds of perceptual motor disabilities, various kinds of conduct disorder, various kinds of minimal neurological findings, etc. It is exactly the confusion which results from such labeling which causes the author to prefer the term minimal brain dysfunction which is often used by pediatric neurologists. This term does not imply brain damage though there may be cases in which hyperactivity is associated with, caused by, or incidental to brain damage.

From the time Bradley introduced the use of amphetamines in the treatment of certain kinds of behavior disorders in children, in the mid-1930's, until the mid-1960's there was a lot of distrust about this kind of medication and this was manifested by the use of the term "paradoxical reaction." It was as if the stimulants had a peculiar reverse reaction and caused the overactive children to be quiet, as a sedative would cause other children to be quiet. Gradually it became recognized as a more reasonable and understandable treatment as the laboratory scientists began to identify brain chemistries and recognized that in certain centers of the brain amphetamine-like chemicals function to permit control and thoughtful awareness.

It finally became well established that portions of the old brain (parts of the old brain which permit animals in the wild to be in control and constantly aware of the environment) function through stimulant chemicals such as dopamine and norepinephrine. With these discoveries it became much more accepted that we were not providing a foreign substance medication to hyperactive children, but rather that we were helping their brain centers to provide appropriate amounts of chemicals which are normal. In this sense, we can say that by the use of stimulant medications for the hyperactive child we are treating a deficiency disease—we are causing the availability of sufficient amounts of dopamine and/or norepinephrine.

The recognition that we were helping the brain centers produce the chemical they are supposed to produce began to alleviate some of the fear that children on amphetamines or on methylphenidate would become addicted with long-term usage. And it has proven to be so that most of the children, if asked "What do you notice when you take the pill?", will say "nothing." If they are then asked, "How is your world going now?" they will suddenly brighten and give answers such as, "My mom likes me now." "I've got lots of friends." "It makes me do my arithmetic."

One of the most difficult parts of dealing with this condition is to recognize that it is either a very, very complex condition or it is several conditions and that these several conditions have the ability to

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overlap each other. Perhaps the laboratory scientists will one day help us to recognize such things as that a defective serotonin dependent brain center may interact in such a way with a dopamine dependent brain center as to make the symptoms appear to be out of the dopamine dependent brain center. In such a case, the use of a stimulant medication might not help and the use of a major tranquilizer might help.

Prevalence: The common estimation is that in any school classroom of twenty children there is one hyperactive child. However, the multitude of forms that this condition takes may ultimately lead us to recognize that there is a higher than 5% prevalence and that a certain classroom which does, indeed, have one hyperactive child in it may also have one or two children with specific learning disabilities which are related to the same condition, but the children show no hyperactivity.

It is well recognized that this condition in all of its forms is much more commonly found in boys than in girls, and there have been claims that the prognosis for girls with hyperkinesis is much less satisfactory than for boys. But this author has not found that to be true.

Length of Treatment: Fifteen years ago it was still considered appropriate to tell parents that all hyperkinetic children would be off medication by the mid-teens. This is no longer appropriate and it is recognized that a considerable proportion go on into their adulthood with what is now called a residual form of attention deficit disorder. There are estimations that 25% go into adulthood still in sufficient trouble that it makes a considerable impact on their lives. Some carry into their adulthood only a tendency to become disorganized under pressure or a little wild if they drink at a party or are not able to sit down and seriously read. The author consistently has a few parents on medication with their children. These are usually women, of course, since it is mostly women who are willing to put themselves into the position of being patients.

Treatment: Something like 75% of minimal brain dysfunction children respond well to stimulant medications. The other 25% require us to consider allergies, peculiar sensitivities to food substances or additives such as artificial flavorings and colorings, the use of anticonvulsant medications, the use of neuroleptics, the treatment of gastrointestinal difficulties which come under the title of celiac disease, and/or sensitivity to glutens. Then we must always keep in mind that these children live in families in which they may experience troubled relationships just like all other children who come to psychiatric attention may have experienced. We have to keep

in mind, too, that the tempermental makeup of the child may make him a misfit with the rest of his family. Those children with troublesome conduct disorders must also have treatment with play therapy, group play therapy and/or family therapy.

The mention of poor parenting techniques causes the author to issue a caveat. Do not assume that the child you see is the result of poor parenting. All too frequently the author has found it necessary to relieve the parents of their own guilt because their physician has told them they have done a bad job and that they are the cause of the child's difficulty.

Dosage of Medication: This is a difficult one. Thinking only of the 75% of children who respond to stimulant medications, we can indicate that there are four primary medications used. Dextroamphetamine (Dexedrine) has been used the longest followed by methylphenidate (Ritalin) which has, by and large, become the most popular. Pemolene (Cylert) appears to be gaining popularity. Certain of the tricyclic antidepressants are also used. The antidepressants were discovered rather accidentally. They were used for bed-wetting children. Bed-wetting is common among hyperactive children, especially boys. It occurred that when antidepressants were used at bedtime to help control the bed-wetting, the mothers would report a change in behavior of the children in the morning so that getting out of bed in the morning was not such a dreadful ordeal. For our purposes we will stick with the use of dextroamphetamine and methylphenidate as we talk about dosage. There is no such thing as a definite dosage. Following the typical pediatric method of milligram per kilogram dosage does not work. It is a matter of trial and error, and that fact alone is very discouraging to many physicians. In two successive appointments on one day, the author saw a fifteen-year-old boy and a seven-year-old boy. The fifteen-year-old boy was on 5 mg of dextroamphetamine each day and the seven-year-old boy was on 40 mg of dextroamphetamine each day.

The author's routine is to start medication at a low dose, such as 5 mg of dextroamphetamine or 5 mg of methylphenidate the first thing in the morning (trying to get Ritalin in at least a half hour before the intake of any milk). These medications work very rapidly so it is possible to have the parent call within three or four days and report any results. The dosage may be increased, and increased, and increased until obvious positive results have been obtained. In most instances the medication effectiveness wears off within about four hours and, therefore, the next step is to start a low dose at noon and gradually increase until beneficial effects have

been reached. This same procedure may be repeated for a four o'clock dose, if necessary.

Side Effects: There are very few side effects to either of these medications used in this condition. Methylphenidate may cause some stomachache for a few days, but that usually passes. There may be some complaint of headache or nervousness for a few days with either of the medications. There may also be a problem which is not so much a side effect as an adjustment disorder when the child really can't understand himself and the parents can't understand what's happening to him either. There is the possibility of a rather confusing circumstance in which, as the dosage is being slowly increased, the child becomes quite emotional and he may even become quite babyish. The suggestion is that the dosage continue to be increased, and the explanation given by this author is that we have the child caught in a position in which he cannot function in his usual way nor has he reached a stage at which he can function in a better way, and thus he is lost in the middle and is frightened and unhappy. The most serious side effect which has been reported is one of lessened speed in growth. This is, indeed, a rare event. The author has seen two such cases. In both instances it was necessary to only stop the four o'clock afternoon dose. There was a growth spurt which brought each child back into the previously recorded growth curve within about three weeks. and then the afternoon dose was restarted because of the devastating effect their behavior had upon their social situation. This stop-and-start was repeated a number of times.

Medication Vacations: This subject is being given a special title and a special paragraph because it is one of the most problematic parts of this whole subject. For those who accept that this treatment method is a treatment for a deficiency disorder, there is only one reason for medication vacations—to see if the child still requires the medication. This thought brings in an additional concept that the minimal brain dysfunction may, in some cases, actually be a function of a developmental delay. The hope is that in one child or another the developmental mechanism will fall into place and the patient's own mechanisms will take over. With this kind of reasoning the author requests a two day medication vacation twice a year. Since most of our patients are school age we request that these medication vacations be at quiet times in the school year, such as just before the usual fall and spring parent-teacher conference. This gives the teacher, the parent, and the child an opportunity to observe the results. If the symptoms return, the medication is restarted.

Since so many of these children have trouble as

much in their social lives as in their school lives, and also since there are many things to be learned in the summer and during vacations which are as important as things to be learned in school, it seems unfair to take the medication away from the child in the summer or other non-school time. Using the argument of a deficiency disease, one could say that it would be ridiculous to take insulin away from a diabetic child in the summertime.

Complications: The author uses this title rather than put this thought under side effects because major complications are in the social environment of the child. Teachers taught according to certain theories may feel offended that a method other than their own is being used. More distant family members may feel that a child is being "drugged" to make him be good. The parents have to be supported in this battle, and they can often win when the doubting persons are subjected to the changes in the child during a medication vacation.

Examples: Just recently a seven year old child and the parents were subjected to considerable criticism by their pediatrician and by his teacher. The problem had to be that they were bad parents. This particular child was not physically overactive. He did talk out loud when it was inappropriate and when it disturbed the other children, and he was not learning anything in school, even though it was suspected that he was potentially at least normal and perhaps better than normal in intelligence. On medication he became so skillful as a student that he was allowed a freedom to study at his own rate. He also became much more conscious of the needs of the other children and no longer interrupted. Then came a medication vacation and the teacher very easily identified that he was off medication. There was an accidental test of this situation in that a few days after medication was resumed the mother forgot to give the child his morning dosage. (He required only the morning dosage.) She remembered her mistake by mid-morning, but decided to do nothing about it, and shortly after three o'clock that afternoon she received a telephone call from the teacher asking what had happened because the child's behavior had completely reverted to his old ways and he was completely unable to study. Thus the teacher was able to see the boy on medication and on a medication vacation which she knew about, and then a medication vacation which was accidental and she knew nothing about. The teacher's response to all of this was to remark to the mother of this child that she had another child in her class that she was going to talk about with the parents.

Example #2: An eight year old girl, very pretty,

very bright, very smiley, and very friendly could not sit still. Her teacher made a path around the periphery of the room and permitted her to walk all day long. She did this without making noise, without tripping over the other kids, without hitting anybody, and without dropping anything. She listened to the teacher; she could handle everything except sitting still. She could be called upon to respond to the lessons being learned and she could respond from whatever position she happened to be in in her tour around the room. This situation lasted most of a school year and everybody was content with it, except that the teacher recognized that this child was going to pass on to another class and she was not going to get the same privileges. It was with this that she was referred. She was able to sit after the appropriate medication dosage was found, and there were no other changes noted. She was still the smiley, friendly, lovely little girl, but now she could also sit patiently and do what was required of her. Incidentally, the family could now take her to church with them.

Example #3: An eighteen month old boy whose family lived in a very small community with houses widespread had the neighbors locking their doors to keep him out of their homes because when he was allowed in the homes he wrecked everything he could get anywhere near. He did not wreck in the sense of anger, he just simply did not seem to be able to stop. He climbed on things and he investigated everything. This mother was able to indicate that this child, who one of three, was recognized by her as being hyperactive even while still in her belly. He kicked much more than his siblings had kicked. He walked at nine months, he ran at nine months, and he was first found climbing on top of the refrigerator at nine months. Fortunately dextroamphetamine comes also in a liquid form, so we were able to modify the dosage appropriate to his size. Within a few days of starting medication the mother reported that he was hiding under the table whimpering like a whipped puppy. She bravely accepted the recommendation that she double the dose. With that first increased dose he became very dull and apparently uninterested in his surroundings. However, the father identified that later that day when he took this child to visit his grandparents he was, for the first time, a joy to be around and he could even play with his little cousins appropriately. This encouraged us to continue to readjust the dosage. The mother finally reported that one of her neighbors asked, "What's the matter with Jimmy, he's being so good?"

Example #4: A nine year old boy with only modest hyperactivity which really hadn't attracted much

attention, but with very severe reading disability, demonstrated many of the findings one ordinarily finds in a soft sign examination. It was decided to try medication, aiming only at the mild hyperactivity and some tendency to fight with friends when playing in the yard. After the dosage was adjusted, he looked at a plant on a table in the house and said, "Hey mom, that thing has leaves all over it." The mother agreed and said that it had always had leaves. He said, "No, it used to be just green, but now it's got leaves." The mother's response was, "And, my God, we expected him to be able to read!" Once he could identify individual letters and individual words on a page, he soon proved to be one of the best readers in his class.

Example #5: This case demonstrates the wide differences among children with the same condition, and at the same time points somewhat at the length of time of continuous medication. A five year old boy was seen by a psychiatrist because he was "a blob." The mother really expected that she was going to be told that her son was seriously retarded. By good fortune she had brought along her three year old son who took the psychiatrist's office apart during the one hour interview. The psychiatrist, judging that both children were suffering the same condition but with different manifestations, started both of the children on dextroamphetamine. Two years later, because of illness on the part of that psychiatrist, these two children came under the care of this author, who maintained their medication control for the next ten years. As the older of the children was just finishing his junior year in high school, and the younger boy was just finishing his sophomore year in high school, it was suggested we try a medication vacation. Almost of a single voice the two boys protested, and their explanation was that they were within a month of their final examinations of that year and there were certian subjects each of them had learned he had real trouble with, if he did not have the medication. It was therefore permitted that they continue the medication until the examinations were over. They then dropped the medications through the summer, and as they went into the next school year they did not resume medications and they had no trouble. The older son went on to a very long, complex, two-year training in work with machines and the younger went on to college. Neither returned to the use of medication. It is the author's speculation that anyone visiting at that home when these two young men are also at home would find that the older one would be out in the garage with his father working on the car saying absolutely nothing and being thoroughly happy and content. Meanwhile, the younger one would be in

the house competing with his mother as to who could talk the most.

Example #6: This will be the last and the most complicated of the cases presented. This concerns a little boy, age seven, black, and one of three siblings, all of whom were adopted. The younger brother was hyperactive. The younger sister was educationally handicapped. The seven year old was brought to our attention because of the extreme conduct disorder. He had neighbors calling their children in if he went out to play. He had the school authorities seriously considering the recommendation that he be sent to a residential treatment facility. He was very active, very argumentative, very aggressive, and was learning nothing. There was no way he could be instructed to follow a direction which was anything but simple. A direction which included, "Brush your teeth and come to breakfast" would be lost because he could not follow two thoughts. It happened that the author's office was on the second floor so we walked from the waiting room to the office and in the process the little boy was found to stumble on every step. Thus, we could add that not only did he have the hyperactivity and learning disability and conduct disorder, he was also physically clumsy. There was nothing in the soft sign neurological examination which he performed correctly. His final medication adjustment was dextroamphetamine 45 mg per day. Part of this was in spansule form so that he did not have to take a dosage at school-but he did take medication before and after school. By the time he was in junior high school he was the star athlete of his school. He was the one to whom the other children turned to settle arguments and disputes. He was an average student. He could appropriately sit for long periods of time and study, or be up and about and be very active as the situation dictated. At the last visit with this author, he came to the office while on medication vacation. Now age thirteen, he stumbled on every step just as he had when he was age seven. He could not study; he could sit still, and he made an obvious effort to be sure that he stayed still. If he was given a three-part instruction, he could not follow it, but he wanted to follow it. He wanted to be cooperative. Upon being restarted on his medication, he once again became the star athlete of his school. He was an average student and a generally good kid.

There are many variations of the foregoing themes, such as the little boy who kept wrecking his bicycle by running it into a gatepost. Everybody thought he was just being nasty and obstinate. After medication was started he wondered about the whole situation, and the only way he could explain it was

that, "They moved the gatepost." There was obviously something wrong with the way he visually perceived space and his own body in it. Then there was the boy who was hyperactive, but who was also in a family in which there was much activity such as camping. He could be on a lake and could not see a mother duck and her ducklings swimming along the shore. On medication, he could see them and enjoy the sight.

Do Other Things Work And Do Other Things Account For Some Of The Cases?: The answer to this is yes, but the numbers are small. A child who had had a long infancy history of diarrhea and colic and restlessness and screaming was much more strictly controlled in intake of glutin products and his hyperactivity quieted. A child who was very hyperactive was tried on everything that we could think of, with no good results until we decided to see what would happen if we modified some of the other brain chemistry. On deanol, which reportedly causes an increase in available acetylcholine, the results as described by a residential treatment center were "magic." A little boy refused candy from the examiner and smilingly explained, "My mom and I found out that if I eat chocolate I get worse." One mother reported that her boy was worse the day after Halloween when he ate all of the trick or treat candies he had obtained the evening before.

One last example can direct our attention to the residual form of this disorder as it manifests itself in the adult. One mother of two children who were on medication for hyperactivity was constantly in trouble with the child protection authorities because of her behavior. She was a very poor housekeeper. She was an impulsive kind of person who might very well just go off and leave her children without protection at night. She frequently went to bars and, as she described it, when she drank she became "rowdy." She also was placed on medication and within a very short time had changed into a very attractive, well-dressed woman who was very capable of taking care of her home and taking care of her children. She stopped going to the bars. She got herself a full-time job, and then registered for college courses at night. The child protection authorities no longer worried about her or the care of her children.

Conclusion and Discussion: This paper is not intended to be all inclusive, but rather is intended to broaden the horizon or the view of the family physician and pediatrician as they work with the families and children suffering with this disorder, or with these disorders, whichever history proves to be correct. It is hoped that we can keep in mind that this condition is more than a problem with attention, unless there is some translation of the word attention

which is, indeed, all inclusive. All possibilities must be kept in mind, including allergies and specific sensitivities to chemicals taken in and to abnormal chemicals produced due to malfunctioning of organs of the body, and to difficulties generated out of malfunctioning with families. All of these, and many others, must be considered.

A relationship which was not emphasized in the body of this article is exemplified by the case of a child with a form of epilepsy who, on medication for the epilepsy, demonstrated an increase in hyperactivity. The sedative effect of the chemicals used for the epilepsy caused the increase in hyperactivity. It was easy to mix the anticonvulsant medications with stimulant medications to control both the seizures and the hyperactivity.

Thus, it is important to remember that this condition may be the result of many things. The poor teaching techniques and the poor parenting techniques may be the result of the extreme difficulty of living with and guiding such children. It is to be remembered also that it is very important to get started in treatment as early as possible so that the child will have the benefit of years of good success instead of the pain of constant failure.

ADDENDUM

Recently it has been suggested that the use of methylphenidate Ritalin for attention deficit disorder may be related in some way to the development of Gilles de la Tourette syndrome or tics. The author has seen two such cases. The cause/effect relationship in the very few cases which have been reported points toward the dopaminergic activity of methylphenidate. It is equally possible that in this very small number of cases, Tourette syndrome is simply running parallel to the attention deficit disorder as a separate disorder. At the present time the answer is to be aware.

REFERENCES

The following listing will be composed largely of easy to read materials, some of which are very appropriate for parents and teachers.

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S Department of Health

Another Radiation Hazard in the Home

During the past few years the use of home computers and computerized games which require some form of video display has swept throughout the United States and South Dakota. Most often the video games are played by children and adolescents and frequently are viewed upon the old, semi-worn out family TV set. Many of the older tube type TV sets, which have an average life span of about eleven years, were manufactured from 1960 until January 15, 1970 at which time emission standards for color TV sets were adopted and enforced. The radiation emitted by these older sets is usually within accepted limits if the set is watched at the usual viewing distance of about 165 cm for children and 250 cm for adults.

In a pungent and thought provoking letter to the editor of the New England Journal of Medicine, Nashel, Korman, and Bowman (1982) presented theoretical data, discussed this matter briefly and have questioned whether youngsters, who are using older color TV sets for video-display screens, are now receiving radiation exposure from the older TV sets which is far in excess of the recommended minimums.

The quantity of radiation received at a given point bears a direct correlation with the distance of the target from the emitting source (inversly proportional to the square of the distance). For this reason, the distance separating the color TV set being used as a video-monitor and the individual viewing the screen is a critical factor determining the total amount of radiation which will ultimately be received by the viewer. In addition, the amount of time spent viewing the TV screen is also an important determinant as to the total amount of radiation received.

During the operation of computers and while playing video games, the user of the apparatus usually is located close to the TV set, often less than 46 cm. This proximity to the radiation source increases radiation exposure drastically, such that the estimated average radiation dose to the thyroid could be 779 mrem per year and the dose to the eyes could be 890 mrem per year (assuming usage of the TV set for 2 hr/day or 730 viewing hours per year). The recommended maximum radiation exposure per year by the National Council for Radiation Protec-

tion and Measurement is 100 merm for persons under 18 years of age.

Although the physicians of South Dakota are not in a position to control this potential health hazard for the people of this state, they are in the capacity of advisors to their patients and their families and can counsel them in regard to this potentially serious health hazard. Hopefully, through the mechanism of applied preventive medicine, future cataracts and thyroid carcinomas may be circumvented.

J. B. Gregg, M.D., Chief Office of Medical Services W. F. Stanage, M.D., Associate Chief Office of Medical Services Joyce Glawe, Radiation Safety Specialist Division of Public Health

REFERENCE

 Nashel, DJ, Korman, LY, and Bowman, JO, Radiation hazard of video screens. NEJ Med, 307:891, 1982.



S Auxiliary News



Available upon request for use by the member will be application and program participation forms, guidelines, and related materials. Also available to the member would be a formal report of all courses attended under the PDS program and a personal letter of commendation from the AMA Auxiliary national president.

Mrs. Richard I. Porter (Marlys)
South Dakota State Medical Auxiliary President

The American Medical Association Auxiliary, Inc., one of the nation's largest volunteer organizations, is establishing a Professional Skills Development Program for volunteers seeking to market their many skills learned, developed, and implemented as a volunteer.

The PSD program would provide a central source for documentation of AMA Auxiliary member participation in instructional courses and training programs which develop new, or enhance the current business and professional skills of the volunteer.

Course offerings which would qualify documentation in PSD include educational programs offered by medical auxiliaries, other volunteer organizations, business and professional groups, and institutions of higher learning throughout the nation.

The following data would be kept on file at national headquarters for members participating in the PSD program.

- -the title of each course taken
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This Is Your Medical Association

Dr. Robert E. Van Demark, **Sr.**, Sioux Falls, has been named Honorary Chairman for the South Dakota Lung Association.

Roscoe Dean, M.D., Wessington Springs, was presented with the Vic Stephans award for his outstanding contributions to emergency medical services. Dr. Dean's part in the establishment of a state-wide emergency medical system began in 1969 when the governor appointed him health advisor to the newly implemented Emergency Health Services. Later that same year, the governor appointed Dr. Dean a member of the committee to implement the Ambulance Committee for the state of South Dakota.

Drs. Lawrence Finney, Earl Kemp, Verlynne Volin and Michael Farritor all of Sioux Falls, Lonnie Waltner of Bridgewater and David Buchanan of Huron have all been recently recertified as diplomates of the American Board of Family Practice.

Several South Dakota physicians who have been recently named Fellows of the American Academy of Family Physicians are Dr. Edward Clark of Sioux Falls, Dr. Clark Likness of Watertown, Dr. David Yecha of Gettysburg and Dr. George Mangulis of Philip.

Dr. Eldon Bell of Webster has been awarded a first place plaque by the state cancer awards committee for professional work contributed to the Day County Cancer unit. He presented a program on cholesterol and cancer.

John Hill, M.D., 87, Yankton, died recently. He was born at Imperial, Neb. and attended medical school at the U. of Pennsylvania and graduated in 1927. He served in the U.S. Army Medical Corps during WWII. He was employed on the staff of the Human Services Center in Yankton from 1930-38. He returned to the center in 1956 and worked there until retiring in 1972.

Survivors include his wife, Alice and one sister, Janet Lamb of Utah.

Rapid City physician, William Mattson, M.D. was elected president of the South Dakota Chapter of the American College of Surgeons at their annual meeting in Chicago, Ill.

Robert H. Quinn, M.D., Sioux Falls, has been appointed vice president for health affairs, dean of the School of Medicine and professor of surgery at the University of South Dakota. He has been serving as acting vice president for health affairs and dean of the medical school. Dr. Quinn has been associated with the U. of South Dakota Medical School since 1947.

Dr. Anthony Petres, Salem, was honored by John Hopkins University when they presented him with a gold watch for reaching his 50th graduation anniversary and being the only member of the class still practicing medicine.

Fred Duimstra, M.D., 77, of Vilonia, Ark., formerly of Sioux Falls, died in Conway, Ark.

He was born in Hull, Iowa and moved to South Dakota as a child. He attended Augustana College and the U. of South Dakota prior to graduating from the U. of Arkansas School of Medicine in 1937. He interned in Little Rock, Ark. He served in the U.S. Army during WWII and in 1946 he returned to Sioux Falls to practice medicine. He retired in 1977 and moved to Arkansas.

Survivors include his wife, Elma; one son, Dwight of Sioux Falls; five grandchildren; two brothers and three sisters.

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S Future Meetings

April

- Advanced Cardiac Life Support, Burlington, IA, April 1-3. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- IX Annual Conference on Perinatal Medicine, Des Moines, IA, April 5-6. AMA Category 1 credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Ophthalomology Clinical Conference, U. of Iowa, Iowa City, IA, April 6. AMA Category I credits. Contact: Richard Caplan,
- Life, Faith, Hope & Magic—The Chaplaincy in a Children's Cancer Center, 8th Annual Pediatric Mental Health Conference, Shamrock Hilton, Houston, TX, April 21-22. Contact: Jeff Rasco, Off. of Conf. Serv., U. of TX, M.D. Anderson Hosp. & Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.
- Medical Documentation—Present and Future Challenges, Sheraton Boston Downtown, Boston, MA, April 22-23. Fee: \$295. Contact: Inst. of Medical Record Economics, 121 Mt. Vernon St., Boston, MA 02108. Phone: (617) 720-2229.
- Advanced Cardiac Imaging, Sheraton-Harbor Island Hotel, San Diego, CA, April 28-30. 18½ hrs. AMA Category I credits. Fee: \$250. Contact: Nomi Feldman, Conf. Cood., 3770 Tansy, San Diego, CA 92121. Phone: (619) 453-6222.

May

- Iowa Medical Society Scientific Session, U. of Iowa, Iowa City, IA, May 1-2. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Obstetrics-Gynecology Postgraduate Conference, U. of Iowa, Iowa City, IA, May 2-3. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Family Practice Review and Update, Radisson Hotel, St. Paul, MN, May 2-6. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Cardiology Today, U. of Iowa, Iowa City, IA, May 3-6. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Otolaryngology Clinical Conference, U. of Iowa, Iowa City, IA, May 13. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Topics and Advances in Pediatrics, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 16-17. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, May 19. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- American Cancer Society National Conference—Breast Cancer—1983, Boston Sheraton Hotel, Boston, MA, May 19-21. 161/2

- hrs. AAFP & AMA Category I credits. Contact: N. G. Bottiglieri, M.D., Breast Cancer Conf., AM. Cancer Soc., 777 Third Ave., New York, NY 10017. Phone: (212) 37I-2900.
- AAMI 18th Annual Meeting and Exhibit Program, Loews Anatole, Dallas, TX, May 22-25. Contact: AAMI, 1901 N. Ft. Myer Dr., Ste. 602, Arlington, VA 22209. Phone: (703) 525-4890.
- Congenital Heart Disease, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 23-24. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Intensive Course in Pediatric Nutrition, U. of Iowa, Iowa City, IA, May 23-27. AMA Category I credits. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Current Concepts in Radiation Therapy, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 25-27. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

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The Assessment of Fetal Lung Maturity With Phospholipid Profiles: The Importance of Phosphatidylglycerol

Clinicopathological Conference Seventeen Year Old Caucasian Male With Recurrent Abdominal Pain

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Clinicopathological Conference Fifty One Year Old Caucasian Female With Persistent Diarrhea



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S Clinicopathological Conference

Seventeen Year Old Caucasian Male With Recurrent Abdominal Pain

Ray H. Krueger, MD* Discusser

John F. Barlow, M.D.**
Editor

Case #877 999 3

This 17 year old Caucasian male entered Sioux Valley Hospital because of recurring abdominal pain.

The patient had been well until 1½ months prior to admission when he developed abdominal pain in the morning and would have to return from school often. The pain was not crampy or associated with nausea or vomiting and was not precipitated by food or any particular initiating factor. There was no gross blood in the stool. The patient's appetite had remained reasonably good. During attacks of the pain, the patient would try to move about to find a comfortable position or become restless and walk. There had been no change in bowel habits. Two weeks prior to admission, a barium enema examination was normal. There was no other past history of hospitalizations or serious illnesses.

PHYSICAL EXAMINATION: Pulse 70/min. and regular, afebrile, respirations 18/min. and regular, blood pressure 108 systolic and 58 diastolic. The patient did not have pain at the time of the examination and was well developed and well nourished. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The heart was not enlarged and there was a regular rhythm with no murmurs. Examination of the abdomen showed slight tenderness in the right upper quadrant with no palpable organs or masses. A rectal examination and proctoscopy examination were unremarkable.

LABORATORY DATA: Urinalysis clear, yellow, specific gravity 1.016,pH 7.0, negative for protein, glucose, ketone bodies, and hemoglobin; sediment—negative. Hemoglobin 10.5 gm/dl, hematocrit 33 vol/dl, red count 4.07 million/mm3 (4.07 \times $10^{12}/L$, mean corpuscular hemoglobin 27 micromicrograms (picograms), mean corpuscular volume 81 cubic micra (fermetoliters), mean corpuscular hemoglobin concentration 32%, total leukocyte count 18,600/mm3 (18.6 \times $10^9/L$) with 78% neutrophils, 12% neutrophilic bands, 1% eosinophils and 9% lymphocytes. The red cells showed slight polychromasia. The platelet count was 446,000/mm3 (446 \times $10^9/L$.) Electrolytes, lactic dehydrogenase (LD), alkaline phosphatase, aspartate aminotransferase (SGOT), in-

organic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid and cholesterol were essentially within normal limits. Total iron 43 ug/dl (35 to 171 ug/dl), total iron binding capacity 478 ug/dl (normal 221 to 481 ug/dl), percent saturation 9% (normal 16 to 36%). Two stools for occult blood were positive. An upper gastrointestinal series and small bowel follow-through showed an abnormal area in the right mid-abdomen with a suggestion of mass effect and central ulceration. A radioisotope scan of the abdomen with technetium pertechnetate revealed an abnormal accumulation in the mid abdomen. An operation was performed.

DR. KRUEGER: In my discussion of the case presented, I will follow the method of Eddy, et. al⁵. The first step in solving this exercise lies in aggregation of the findings presented. They are as follows:

- 1. History of abdominal pain for one and one half months in a 17 year old man.
- Physical examination which is unremarkable except for tenderness in the right upper quadrant.
- 3. Normal laboratory findings including normal plasma electrolytes, normal liver function tests and other chemistries and normal urinalysis.
- Microcytic anemia suggesting mild to moderate iron deficiency. This was associated with gastrointestinal blood loss as evidenced by the finding of occult blood in the stool.
- 5. X-ray studies including upper gastrointestinal series with small bowel follow-through showing an abnormality in the right mid-abdomen, producing a mass effect with possible central ulceration and a normal barium enema examination of the colon.
- A technetium pertechnetate scan of the abdomen revealing an abnormal accumulation in the midabdomen. This abnormal uptake was seen at the

^{*} Resident in Family and Community Medicine, Sioux Falls, SD. University of South Dakota, Affiliated Program.

^{**}Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

same time as uptake in the gastric mucosa of the stomach.

- 7. An elevated white count with a shift to the left. The pivotal finding was the abnormal radioisotope scan of the abdomen. Causes of this abnormality according to Berquist et al.⁶ and Goel⁴ include:
 - 1. Meckel's diverticulum.
 - 2. Small bowel obstruction.
- 3. Intussusception
- 4. Hydronephrosis
- 5. Arterial venous malformation.
- 6. Barrett's esophagus
- 7. Ectopic gastric mucosa in small bowel.
- 8. Duodenal ulcer.
- 9. Intrathoracic gastrogenic cyst.
- 10. Retained gastric antrum.

To prune down this list, one can eliminate Barrett's esophagus as well as an intrathoracic gastrogenic cyst because the uptake is in the wrong area.

The patient has no history of gastric surgery and this retained gastric antrum would not be possible. A duodenal ulcer would be unlikely with a negative upper gastrointestinal series. Additionally, the patient's pattern of pain would be somewhat unusual for duodenal ulcer. A small bowel obstruction secondary to intussusception would likely occur in a younger patient and would not have such a drawn out history of abdominal pain. Additionally, the upper gastrointestinal series did not show an intussusception. An intussusception in this age group could occur with a polyp of Peutz-Jehgers syndrome. An arterial venous malformation has been suggested in the literature to be one cause of a false positive scan for Meckel's diverticulum. However, in the experience of Berquist⁶ at the Mayo Clinic these malformations do not usually cause a positive scan. Hydronephrosis or other urinary tract abnormality would not explain the patient's blood loss.

Thus, the main diagnostic possibility remaining given the patient's positive radionuclide scan and characteristic history is Meckel's diverticulum. I suppose an ectopic patch of gastric mucosa in the small bowel unassociated with Meckel's diverticulum is possible, but this is rare. The gastrointestinal blood loss is a prominent part of this patient's clinical picture and certainly blood loss is most common complication of Meckel's diverticulum. Gastric mucosa is present in 50% of the cases of Meckel's diverticulum and is said to be present in virtually all cases that involve bleeding. The gastric mucosa secrets hydrochloric acid which is responsible for ileal ulceration with subsequent bleeding. The next most common complication of a Meckel's is intestinal obstruction which presents usually as intussusception with the diverticulum as the leading edge.

This usually presents as an urge to defecate with current jelly stool. Diverticulitis of a Meckel's diverticulum can occur. This complication presents a picture similar to appendicitis but, of course, can have variation in location of pain due to the position of the diverticulum. More unusual complications involving a Meckel's diverticulum include lipoma, fibroma, angioma, leiomyoma, carcinoid, adenocarcinoma and sarcoma. Additionally bands, knots, volvulus, enteroliths, foreign bodies, parasites or regional enteritis may complicate Meckel's diverticulum. The patient's elevation of white count with the shift to the left may indicate an element of diverticulitis in his presentation, although lack of more systemic symptoms and the fact that the patient was not having pain at the time of surgery would make this a bit less likely. One might theorize also that perhaps a tumor might be associated with this patient's condition, accounting for the mass effect and central ulceration noted on the upper gastrointestinal series.

Meckel's diverticulum is a true diverticulum arising from the antimesenteric border of the ileum. It is composed of a remnant of omphalomesenteric duct which is usually obliterated by the seventh week of fetal development. The incidence varies from 0.3 to 2% in the general population but it has been higher in some surgical series. Routine removal of Meckel's diverticulum has been advocated by some if found incidental at laparotomy. According to Soltero and Bill this is probably not indicated given the usual benign natural history of Meckel's diverticulum and the possible morbidity associated with incidental removal. Removal, however, is indicated when it is suspected to be a cause of abdominal symptoms.

Dr. Krueger's Diagnoses Meckel's Diverticulum

DR. BARLOW: I would first like to show the scan and x-rays. The abnormal area mentioned in the protocol on the radionuclide scan is in the midabdomen above the bladder (Figure 1). The basic principle behind this type of procedure is that technetium pertechnetate obtained directly from a molybdenum generator and injected intravenously localizes in gastric mucosa. The stomach is normally nicely outlined and abnormal locations of gastric mucosa such as occur in up to half of the cases of Meckel's diverticulum will show abnormal uptake. This, of course, implies that unless there is ectopic gastric mucosa in a Meckel's diverticulum it will not be detected. Dr. Krueger has indicated that there are causes of false-positive results in this study. However, the technique is both non-invasive and simple to perform and is worthwhile if positive. An

unusual correlation in this case is the abnormal x-ray (Figure 2) which shows a mass effect with central abnormality interpreted as an ulceration. I will now let Dr. Ensberg discuss what he found in surgery.

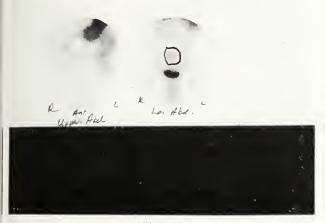


Figure 1
Circled area of technetium pertechnetate abdominal scan is abnormal area of uptake. The prominent area of uptake below the circled area is bladder and at upper right is uptake in gastric mucosa.



Figure 2
Small bowel follow through showing central inverted Meckel's diverticulum with surrounding mass effect.

*DR. DORRENCE E. ENSBERG: Approximately 2 feet from the ileocecal valve there was a gross abnormality in the small bowel. It appeared to be a Meckel's diverticulum folded down along the side of the bowel and mesentery. There was some mild dilatation of the bowel above, but no marked obstructive changes were seen. A small bowel resection with an end-to-end anstomosis was performed because the diverticulum could not be resected locally. Small bowel resection in Meckel's diverticulum is also indicated if small bowel ulceration is present adjacent to the diverticulum. The remainder of the abdominal exploration was unremarkable.

DR. BARLOW: Viewing the resected specimen from the outside a definite abnormality with a protrusion along the serosal surface was seen. On opening the bowel, it became apparent that what was present was a Meckel's diverticulum which had partially inverted (Figure 3). Comparing the gross appearance with the specimen, there is excellent correspondence with the x-ray (Figure 2). The central area interpreted as ulceration was probably a central partially inverted portion of the diverticulum as seen (Figure 3). On microscopic examination marked fibrosis of the diverticular wall was noted. There was ectopic gastric mucosa. No ulceration was noted. There was moderate inflammation and regenerative activity in the adjacent mucosa which could have represented a healed erosion or ulcer.



Figure 3
Gross picture of mucosal surface with inverted Meckel's diverticulum corresponding to x-ray (Figure 2).

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FINAL ANTOMIC DIAGNOSES MECKEL'S DIVERTICULUM WITH PARTIAL INVERSION

I would like to make some final comments on methods of detection of gastrointestinal bleeding by radionuclide techniques in addition to the scan for Meckel's diverticulum which has been described above. Localization of gastrointestinal bleeding in the upper gastrointestinal tract by barium studies or endoscopy is often quite accurate but localization of lower gastrointestinal bleeding is still a problem. Arteriography is certainly a gold standard but a less invasive study is often indicated. There are two radionuclide techniques, one utilizing technetium sulfur colloid and the other using an intravascular agent, technetium labeled red blood cells.

The principle of the technetium sulfur colloid scan for gastrointestinal bleeding is that the injected material is extravasated through the bleeding site and pools at that point. This material is the usual agent for liver and spleen scanning and is trapped in the reticuloendothelial system of these organs. Contrast then develops because the injected material is concentrated in the liver or spleen and extracted from the vascular compartment. This study may be repeated multiple times, shows good localization and does not have interference by vascular structures. It will detect both venous and arterial bleeding. Another advantage is that the technetium scan is easy to perform and the material used is available in most nuclear medicine departments.

The second technique employing an intravascular agent, technetium labelled red blood cells, is advocated by some authors. An intravenous injection of tin followed by external labeling of red blood cells in a heparinized saline solution in a syringe is given. This insures more completely labeled material will circulate for some hours detecting bleeding which is intermittent. Localization appears excellent but may be faulty if the material extravasated into the bowel is propelled forward by peristalsis.

*DR. EARL KEMP: What was the cause of the pain?

DR. KRUEGER: It may have been due to ulceration. Pain is an uncommon presentation in Meckel's diverticulum. Intussusception intermittently or inversion could have produced this. The cause of the pain in Meckel's diverticulum is not always known. I might add that Meckel's diverticulum can also present as iron deficiency anemia.

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SPresident's Page



We have become accustomed to reading about major articles about to appear in the NEW ENG-LAND JOURNAL OF MEDICINE. These are previewed or summarized in the newspapers. Usually they appear in the larger newspapers, but sometimes the local papers pick them up from the national news services. A disappointing thing about this practice is that we are also accustomed to newspaper stories about "breakthroughs" that never quite materialize. Another disappointing thing about this practice is that other journals are thinking of following it. Apparently, the aim is to give the newspapers the idea that this is a truly prestigious journal—one in which most of the latest advances are reported first. My personal reaction is to feel cheated. I subscribe to the NEW ENGLAND JOURNAL OF MEDICINE so I will know about these advances as soon as possible.

The popular press was probably a factor in the

Senate defeat of the bill which would have kept the FTC from expanding their turf to the learned professions. The majority of the press on the East Coast was in full cry in this case, and in some cases, the facts weren't allowed to stand in the way of a good story. At least one of our Senators alluded to the fact that the news articles were important to his thinking on this bill.

It seems we find ourselves in an adversary position with the popular press much of the time. It seems to me this is not as true in smaller towns and very much more true in larger cities.

As members of the South Dakota State Medical Association, all of you are important in this matter. By maintaining contact with local newspaper people, you are in a position to correct misstatements of fact and counteract the influence of the big city daily papers.

This is an important part of maintaining our freedom.

Durword M. Laugmo

Durward M. Lang, M.D., President South Dakota State Medical Association

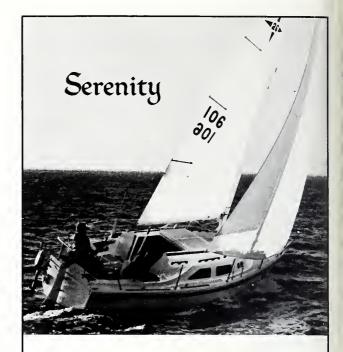
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S Medicine

The Assessment of Fetal Lung Maturity With Phospholipid Profiles: The Importance of Phosphatidylglycerol

David W. Ohrt, M.D., Ph.D.*

ABSTRACT

This is a case of a young female diabetic who gave birth to an infant with respiratory distress syndrome in spite of a mature lecithin:spingomyelin ratio. This paper will discuss in a question-answer format various aspects of amniotic fluid phospholipids and the role of phosphatidylglycerol in predicting fetal lung maturity.

INTRODUCTION

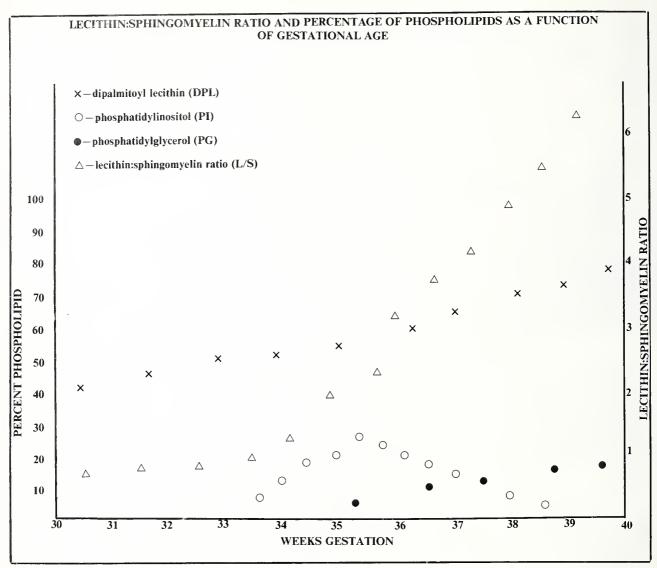
Case Presentation: The patient is an obese 25 year old $G_3P_2AB_0$ juvenile onset insulin dependent diabetic who was admitted to the hospital for an elective repeat C-section. She is 36 weeks gestation by dates and has recently demonstrated a marked increase in uterine size as well as poor control of her diabetes. The patient suffers from mild diabetic retinopathy and mild peripheral neuropathy. Two years ago, she delivered twins, one of whom developed respiratory distress syndrome and required hospitalization for approximately two weeks.

Physical examination revealed a gravid obese young white female weighing 190 pounds. Her blood pressure, temperature, pulse and respiration were; 116/48, 97.6°F, 104/min and 22/min respectively. The uterine fundus was firm with detectable fetal movements and fetal heart rate of 132 beats per minute. A CBC and urinalysis were within normal limits. At the time of hospitalization there were numerous blood sugars which varied from 72 mg/dl to 254 mg/dl. The patient demonstrated symptoms

of hypoglycemia at the lower glucose levels. An amniocentesis was performed and the amniotic fluid subjected to phospholipid studies for fetal lung maturity. Her total lecithin:sphingomyelin ratio and acetone precipitate lecithin:sphingomyelin ratio were 7.8 and 6.3 respectively. Phosphatidylglycerol was absent.

Because of the inability to control her brittle diabetic tendencies, she was brought to the operating room where a low anterior vertical caesarean section was performed. A 4500 gram male fetus was delivered with a Apgar score of 7/10 and 9/10 at 1 and 5 minutes respectively. Respirations were spontaneous and the neonate was brought to the normal newborn nursery in good condition. After several hours the neonate became cyanotic and began requiring increased FIO₂. After 15 hours the infant was maintained on 100% oxygen with an arterial PO₂ of 65 millimeters in accordance with respiratory distress syndrome. X-rays demonstrated the diffuse "ground-glass" pulmonary pattern of this disorder. Gradually there was improvement in the infant's respiratory status and after 14 days in the intensive care nursery, the baby was discharged.

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Question: What are the amniotic fluid phospholipids? From where do they originate?

Answer: The amniotic fluid phospholipids refer to a group of closely related compounds derived principally from the Type II pneumocyte of the fetal alveolar epithelium. These phospholipids make their way to the amniotic fluid by means of the normal amniotic fluid circulation involving a tracheal fluid flow of 1-2 mls. per minute which is principally derived from the pulmonary capillary. Lowering of the surface tension to prevent alveolar collapse during the respiratory cycle is the ultimate function of the phospholipids. 40-70% of the phospholipids consist of dipalmitoyl lecithin (DPL) which is the most surface active of the entire group. Sphingomyelin (S), phosphatidylinositol (PI), phosphatidylethanolamine (PE), phosphatidylserine (PS), phosphatidylglycerol (PG), and others comprise the remainder.² A minor but significant proportion are derived from sources other than the fetal lung. A variety of different lecithins are present in fetal skin,

Figure 1

fetal urine, placenta and fetal gastric mucosa. Furthermore, lecithins are present in maternal blood and could conceivably reach the amniotic fluid. Because both lecithin (L) and sphingomyelin (S) are important components of the cell membranes, these phospholipids are essentially ubiquitous. None of the extrapulmonary lecithins possess a surface activity as powerful as DPL.³

Question: Do the concentrations of the different amniotic fluid phospholipids vary with gestational age? Answer: Yes, the phospholipid composition of the amniotic fluid is a function of gestational age reflecting the degree of maturation of the pneumocyte Type II (Figure 1). Prior to 33 weeks gestation L and S are present in equal quantities. At 33 weeks, the metabolic pathway leading to production of DPL increases resulting in a continued, nearly linear elevation of the L:S ratio until term. PI begins to rise during the third trimester, reaches a maximum at 35 weeks, and then steadily declines as term is approached. PG appears as early as 35 weeks, increases

towards term, and indicates complete maturation of the alveolar surfactant complex. PG and PI tend to provide an added degree of alveolar stability at low lung volumes. Sremains essentially level throughout pregnancy which allows the expression of L in terms of a ratio. PE and PS are of minor compositional significance. It is significant that only trace quantities of PG are seen in body tissues other than lung.

Question: What are some of the rapid tests for assessment of amniotic fluid surfactant? Currently, what is the best method for assessing phospholipids and fetal lung maturity (FLM)?

Answer: Because there are a bewildering number of qualitative chemical techniques for assessing amniotic fluid surfactant, only the more well known methods will be considered. The OD-650 is a well known test which takes advantage of the turbidity produced by phospholipids in an aqueous environment at 650 nm. An absorbance of a 0.15 AU or greater indicates FLM. The major advantage of this test is the rapidity of analysis. The major disadvantages include an occasional false positive prediction of FLM as well as the large number of false negative predictions of respiratory distress syndrome (RDS) for absorbances below 0.15. In other words, many fluids with an OD-650 below 0.15 are in reality mature.

The foam stability test is much like the OD-650 in terms of efficacy, however, it is critically dependent upon technique. In this test a small quantity of amniotic fluid is mixed with ethanol for a given period of time. The presence or absence of foam at the margins of the meniscus is assessed at a defined period of time after mixing. The development of a stable foam after shaking indicates the presence of sufficient surfactant activity and correlates with FLM. As with the OD-650 the foam stability test suffers from an inappropriately high number of false negatives and false positives.⁷

A quite new approach to the evaluation of amniotic fluid surfactant activity involves the measurement of viscosity and is referred to as fluorescence microviscosimetry. As the quantity of phospholipids increase with gestational age, the viscosity decreases. If the viscosity is below some carefully predetermined level, the fetal lungs are assessed as mature. This method summates the total surfactant activity of the fluid and is not specific for any particular phospholipid or phospholipids. The main advantage of this approach is the rapidity of analysis.⁸

Gluck first described a semi-quantitative approach in 1971 for assessing L and S in amniotic fluid. After precipitation of the phospholipid by means of cold acetone, separation of L and S was accomplished by thin layer chromatography and the quantities expressed in terms of the L:S ratio. The best current methodology still involves the use of thin layer chromatography, however, it has been expanded and improved. Not only are L and S assessed in the analysis but other phospholipids including PI and PG can be demonstrated as depicted below in Figure 2.

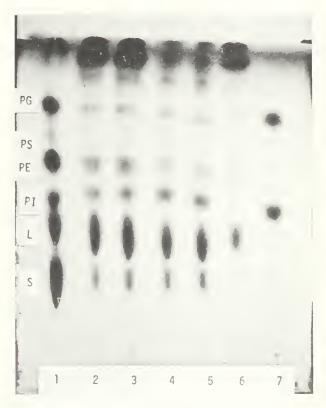


Figure 2
Phospholipid profile by one-dimensional thin layer chromatography

As shown above, each amniotic fluid study involves seven individual determinations. Studies #1 and #7 are controls used for identification and the demonstration of appropriate resolution of phospholipids. Sample #6 represents the acetone soluble fraction remaining in the supernatent after precipitation of cold acetone insoluble lipids. Recently the acetone soluble fraction has been replaced by a phospholipid control. Studies #2 and #3, #4 and #5 represent total phospholipids and acetone precipitated phospholipid respectively. The indicated duplication of each type (total and acetone precipitable phospholipids) are performed for precision evaluation. On the left the individual phospholipids are labeled. Quantitation of the density of L and S allows calculation of the L:S ratio. The presence or absence of PG is assessed visually.

The phospholipid profile by thin layer chroma-

tography is the most reliable assessment of fetal lung maturity. Unlike the previously described tests, phospholipid profiles require several hours to perform as well as special training on the part of the technologist.

Question: What is the difference between total and acetone precipitable lecithin?

Answer: As a result of Gluck's original work, it was pointed out that cold acetone precipitation caused precipitation of mainly DPL. Lecithins from other sources such as fetal skin, fetal urine, and maternal plasma do not precipitate to any significant extent. For this reason, the acetone precipitate is felt to be more representative of the true surface active lecithin. Determination of total phospholipids does not involve a precipitation step and does represent all detectable amniotic fluid phospholipids. Increased quantities of lecithin from extra-pulmonary sources in the total phospholipid studies is the major difference. In accordance with this, the total L:S ratio is always higher than the acetone precipitate L:S ratio. An L:S ratio of 3:1 and 2:1 or greater for total and acetone precipitable phospholipids respectively, suggests FLM in a normal pregnancy. By performing a total and an acetone precipitate an internal check of the validity of the determination is made.9

Question: Is the L:S ratio a reliable predictor of FLM in cases of maternal diabetes and other fetomaternal diseases?

Answer: For normal pregnancies (no evidence of fetal maternal disease), the L:S ratio may falsely predict FLM in about 2% of cases. With complications of feto-maternal disease the false prediction of maturity climbs to 5% or greater. This problem is best exemplified in cases of maternal diabetes since as many as 28% of neonates born to diabetic mothers with insulin dependent diabetes develop RDS inspite of a mature L:S ratio. The mechanism for these false predictions is unknown, however, it may be related to hyperinsulinism in the neonate. Also a variable progression of the L:S has been demonstrated in maternal diabetes. There may be a delay or an acceleration in the progression of the L:S ratio for White diabetic Classes A-C and D-R respectively.14

Although diabetes is the major maternal disease of concern, there are many other feto-maternal complications that have been shown to alter the reliability of the L:S ratio. Disorders such as premature rupture of the membranes, pre-eclampsia, chronic hypertension, cardiovascular disease, hemoglobinopathies, and certain congenital malformations tend to accelerate the development of a mature ratio. On the other hand, renal disease, hepatitis, hydrocephalus, collagen disease, syphilis, and tox-

oplasmosis delays progression. One might expect the L:S ratio to be less reliable under such circumstances since the disease process itself may be altering the ratio independently of biochemical processes leading to surfactant production. Because such variations decrease the reliability of the L:S ratio, there is a definite need to evaluate the presence or absence of PG. Originally these variations with feto-maternal diseases were apparent because the early L:S studies were performed and based upon a predominantly healthy maternal population.¹⁵

Question: How can predictions of fetal lung maturity and respiratory distress syndrome be improved when feto-maternal disease is present?

Answer: PG production by the alveolar type II pneumocyte marks the final maturation of the phospholipid surfactant complex. PG, by lowering the surface tension yet further, adds greater stability to the alveolus at low lung volumes.

Although PG is subject to the same variations in terms of retardation and acceleration of its appearance, its presence nevertheless provides the added assurance of a safe delivery irrespective of the L:S ratio. The absence of PG is a biochemical marker of RDS. The phospholipid profile allows an assessment of the presence or absence of PG along with the determination of both total and acetone precipitate L:S ratio. ¹⁰⁻¹⁵

Question: Are phospholipid profiles affected by blood and meconium contamination?

Answer: The adverse effects of the two most common contaminants encountered in FLM testing, blood and meconium, are well established. The L:S ratio for serum or plasma varies from 1.3 to 2.3. Therefore, an immature L:S ratio may be increased by blood contamination and conversely a higher ratio may be lowered. In the presence of PG, blood does not preclude on accurate prediction of FLM since it is not present in this body fluid. The L:S ratio cannot be interpreted in the presence of meconium. It has been reported that meconium does not affect an assessment of PG.10 Currently the author of this issue is investigating this potential problem and initial studies reveal an intense variably migrating component that can masquerade as PG. This can potentially result in a false positive identification of its presence. Therefore, phospholipid profiles containing meconium must be interpreted with caution. Possible means of circumventing this problem are currently under investigation.

Question: In cases of premature rupture of the membrane, can a phospholipid profile be performed on vaginal specimens? Answer: The best amniotic fluid specimens are obtained by the transabdominal route. In cases of premature rupture of the membranes, vaginal specimens of amniotic fluid are acceptable for phospholipid analysis provided that the fluid is free flowing. Usually the L:S ratio tends to be higher in vaginal specimens which may be due to bacteria. PG can be reliably evaluated, however it is susceptible to dilutional effects by secretions possibly resulting in a false negative assessment. This effect can be minimized by sampling free flowing fluid.^{17,18}

Question: In the case of twin pregnancies with two gestational sacs, is the amniotic fluid of one sac also representative of the other?

Answer: No, the phospholipid profiles of the two sacs may differ markedly. The fluid from one sac may be clearly mature and the other immature. Amniotic fluid specimens should be obtained from each gestational sac if preterm delivery is being contemplated.

Question: How are phospholipid profiles interpreted? Answer: The interpretation is based upon the L:S ratio and the presence or absence of PG. The conclusive assessment is modified by a consideration of the effect of feto-maternal disease. Generally, interpretations fall into one of the five categories below:

- A. Immature Pattern: Total and acetone precipitate L:S less than 2:1 and 1.5:1 respectively. PG absent.
- B. Transitional Pattern: Total and acetone precipitate L:S between 2-3 and 1.5-2.0 respectively.
- C. Mature pattern provided there is no evidence of diabetes or other feto-maternal disease: Total and acetone precipitate L:S at or greater than 3:1 and 2:1 respectively, PG absent.
- D. Mature pattern: Total and acetone precipitate L:S greater than 4:1 and 3:1 respectively, PG present.
- E. Mature pattern with delayed progression of L:S ratio: Total and acetone precipitate less than 4:1 and 3:1, PG present.

The well defined criteria given above is subject to modification depending upon the assessment of component resolution and the presence of possible contaminants. Respiratory distress has been reported for acetone precipitate L:S ratios greater than 2:1 in apparently normal gestations when PG is absent.²⁰ Irrespective of whether the pregnancy is complicated or uncomplicated, there is now a tendency by some obstetricians to wait until PG appears before preterm

delivery, if the clinical situation allows. The logic of such an approach would be strengthened by statistical data designed to assess whether PG occurs in the latter weeks of every gestation. General experience suggests that it does occur in the majority of gestations but numerical data is not available in the literature at this time.

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BRIEF SUMMARY PROCAROIA® CAPSULES

For Dral Use

(Infedipine)

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (Infedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or oroonary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasical component but where vasical components are the component of the control of the control of the component of the control of the con where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vaso-spasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates

or who cannot tolerate those agents.
In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are

incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available infor-mation is not sufficient to predict with confidence the effects of concurrent treatment, especially in

mation is not sufficient to predict with confidence the effects of concurrent freatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.) CONTRÁINDICATIONS: Known hyperesnsitivity reaction to PROCARDIA. WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PRDCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PRDCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose lentanyl appears to be due to the combination of PRDCARDIA and a beta blocker, but the possibility that it may occur with PRDCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic

Increased Angina: Decasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand

associated with decreased distolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PBOCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for

such an event

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vaso-dilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Orug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PRDCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional

of PRDCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart

Interactive reports suggesting fund the confination may increase the inseminou of conjective heart failure, severe hypotension or exacerbation of angina.

Long-acting intrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PRDCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two yournevers in three patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PRDCARDIA to avoid possible over-or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nitedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human december.

man dose.

Pregnancy: Category C. Please see full prescribing information with reterence to teratogenicity in

regimency Category 6. Please see for prescribing information with reterritor to relatogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys. **AOVERSE REACTIONS:** The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, and flushing each occurring in about 10% of peripheral edema, nausea, weakness, and flushing each occurring in about 10% of peripheral edema, nausea, and flushing each occurring in about 10% of peripheral edema, nausea, weakness, and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of peripheral edema, nausea, headache and flushing each occurring in about 10% of peripheral edema, nausea, headache and flushing each occurring in about 10% of peripheral edema, nausea, headache and flushing each occurring each edema, nausea, headache edema, nausea, headache edema, nausea, headache edema, nausea Syncopa episouse and in the etra will reduction in the tube of PRDCAMIDA of concominant annuary ginal medication. Additionally, the following have been reported muscle cramps, nervousness, dyspinea, nasal and chest congestion, diarrhea, constipation, intlammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, purities, uriticaris, ever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension. In addition, more serious adverse events were observed, not readily distinguishable from the nat-

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in lewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGDT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease affer about eleven months of nitedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PRDCARDIA therapy, has been reported twice in the extensive world literature

HDW SUPPLIED: Each orange, soff gelatin PRDCARDIA CAPSULE contains 10 mg of nifedipine PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."



"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA^[*] as soon as it became available. The change in my condition is remarkable."

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks, taking fewer nitroglycerin tablets, doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).

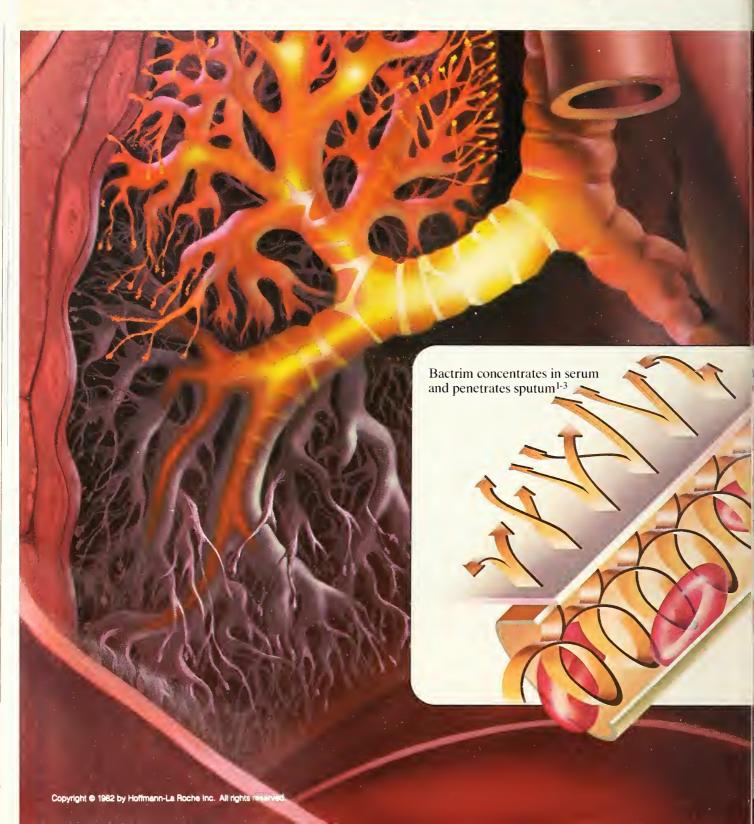


for the varied faces of angina

- *Procardia is indicated for the management of:
 - 1) Confirmed vasospastic angina.
 - 2) Angina where the clinical presentation suggests a possible vasospastic component.
- 3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.



Bactrim attacks the (trimethoprim and sulfamethoxazole/Roche) in acute exacerbations





najorpathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

In sputum cultures from patients with acute exacerbations of chronic bronchitis, H. influenzae and S. pneumoniae are isolated more often than any other pathogens. 4.5 One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.5

Bactrim is effective in vitro against most strains of both S. pneumoniae and H. influenzae—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.6

Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin q.i.d., Bactrim DS proved equally effective on all clinical parameters.7.9 Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of b.i.d. dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

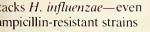
Bactrim also proved more effective than tetracyclines in 10 clinical trials

involving nearly 700 patients. 10 Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

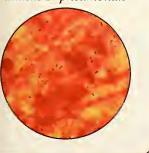
Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: Adv Antimicrob Antineoplastic Chemother I/2:1105-1106, 1971. 2. Jordan GW et al: Can Med Assoc J 112:91S-95S, Jun 14, 1975. 3. Beck H, Pechere JC: Prog Antimicrob Anticancer Chemother 1:663-667, 1969. 4. Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts; Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12 5. Schreiner A et al: Infection 6(2):54-56, 1978. 6. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 7, Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a doubleblind crossover clinical trial, in Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit., pp. 15-16. 8. Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim 14) and ampicillin in the treatment of bronchitic exacerbations. Ibid., pp. 17-18. 9. Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. Ibid., pp. 19-20. 10. Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. Ibid., pp. 13-14.





attacks S. pneumoniae



Economical b.i.d.

BactrimDS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

Bactrim

(trimethoprim and sulfamethoxazole/Roche)

Batora prescribing, plaasa consult complate product information, a aummary of which

Indications and Usage: For the traatment of urlnary tract Intactiona due to auaceptible strains of the following organisms: Escherichia coli, Kiebsielia-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii. It la recommended that initial epiaodea ot uncomplicated urinary tract intections ba treated with a single effective antibacterial agent rather than tha combination. Note The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections For acuta otitis madia in children due to ausceptibla strains of Haemophilus Influenzae or Streptococcus pneumoniae whan in phyaician's judgmant it offara an advantaga over other antimicrobials. To date, there are limited data on the aafety of repeated

usa of Bactrim in childran undar two years ot age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acuta axacarbations of chronic bronchitia in adulta due to auaceptible atraina ot Haemophilus influenzae or Streptococcus pneumonise when in physician's judgment it offera an advantage ovar a single antimicrobial agent.

For enteritia due to suaceptible atraina of Shigelia flexneri and Shigelia sonnei when antibacterial therapy is indicated.

Also for the traatmant of documentad Pneumocystis carinii pnaumonitla.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing motners because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL

PHARYNGITIS. Clinical studies show that patients with group A β -hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis', aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with pur pura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended, therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfa-

methoxazole may interfere with folic acid metabolism, use during pregnancy only if poten-

tial benefits justify the potential risk to the fetus. **Adverse Reactions:** All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Allergic reactions: Erythema multiforme, Stevens Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. Gastro intestinal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis diarrhea, pseudomembranous colitis and pancreatitis. CNS reactions: Headache, peripheral neutritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients, cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies

Dosage: Not recommended for Infants lass than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults. Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shinellosis

Children Recommended dosage for children with urmary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided

doses for 10 days. Use identical daily dosage for 5 days for shigellosis. For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS

Usual adult dosage. 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp (20 ml) b.i.d. for 14 days
PNEUMOCYSTIS CARINII PNEUMONITIS

Recommended dosage. 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for iggested children's dosage table

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100, Tel-E-Dose* packages of 100; Prescription Paks of 20 and 28 Tablets, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 40. Pediatric Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint)



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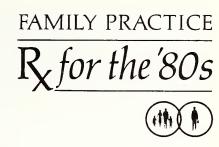
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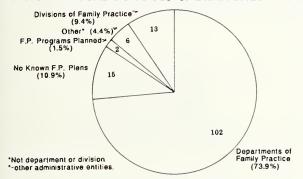
S Chapter News





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

FAMILY PRACTICE PROGRAMS WITHIN THE 138 U.S. MEDICAL SCHOOLS & BRANCHES - 1982



The American Academy of Family Physicians

National Commission/Committee Activities

Secretary-Treasurer L. H. Amundson, M.D. was re-appointed to a second three year term on the AAFP Commission on Education. This commission meets twice yearly and deals with undergraduate and graduate medical educations issues as they impact on the AAFP and the practicing family physician.

Delegate R. W. Friess, M.D. was appointed to a one year term on the new AAFP Committee on Hospitals. This activity was formerly handled by the Commission on Health Care Services, but increased concerns regarding hospital costs, hospital involvement in ambulatory care, and hospital privileges for family physicians led the Congress of Delegates to form this separate body to deal with these timely issues. Committees meet once or twice a year.

'FP in Hospitals' available free to AAFP members

"Family Practice in Hospitals"—a new Academy publication dealing with hospital privileges and clinical family practice departments—is currently available free of charge to Academy members.

The publication was developed by the Subcommittee on Hospitals of the Commission on Health Care Services. It is designed to help answer questions about clinical privileges, establishing clinical departments of family practice, avoiding hospital privilege conflicts and managing conflicts when they occur

conflicts and managing conflicts when they occur.

A copy of "Family Practice in Hospitals" can be obtained by writing the AAFP Order Department, 1740 W. 92nd St., Kansas City, Mo. 64114, or calling on the Academy's toll-free number, 1-800-821-2512.

Physicians who are not Academy members also can obtain a copy of the book for \$5.

1983 Black Hills Summer Seminar

Program Director and SDAFP President-Elect L. W. Finney, M.D. has announced the content for the 1983 Annual Meeting Scientific Seminar, scheduled for Rapid City August 11-13, 1983. The program will have components in Forensic Medicine, Agricultural Occupational Medicine and Psychiatry. J. H. Talley, M.D. of Grover, NC, a family physician speaker at the 1982 national meeting in San Francisco, will be one of the speakers.

National AAFP President Gerald Gehringer from New Orleans will be an invited guest, and give an AAFP update during the Thursday noon luncheon.

Sioux Falls Family Practice Residency Program Anniversary Reunion

James A. Oakland, M.D. of Sioux Falls has announced plans for a Tenth Anniversary Reunion of the Sioux Falls Program, to be held during the 1983 Black Hills Summer Seminar in Rapid City August 11-13, 1983.

Founding Director L. J. Sweeney, M.D. of Sun City, AZ is expected to be present for this event, commemorating the July, 1973 start of the program. Effective June 30, 1983, sixty-six graduates will be recognized.

Graduates are asked to contact James A. Oakland, M.D., 1621 South Minnesota Ave., Sioux Falls, SD 57105, phone 605-336-1015 for further reunion details.

AAFP Research Workshop To Emphasize Practical Approach

You too can be a researcher—without ever leaving your office practice. Conducting research can pay big dividends in personal satisfaction and, at the same time, help solidify the specialty of family practice. In fact, without a firm base of practice-based research, family practice could fall short of its potential.

You can explore the possibilities conveniently at an AAFP weekend seminar scheduled for May 13-15 in Columbus, Ohio. By the end of the second day of the Regional Research Workshop for Family Physicians, you will know enough to make an informed decision about future involvement in research projects. Faculty members will orient participants to each step of the process—designing the project, conducting the literature review, collecting data and publishing conclusions—in plenary session lectures and small group working sessions.

Tuition for the workshop, which is designed by the AAFP Committee on Research and acceptable for 15 hours of Prescribed credit, is \$100.

For additional information and registration materials, contact the AAFP Research Division on the toll-free line, 1-800-821-2512.

Chief, Ambulatory Care Services

Opening for Chief, Ambulatory Care Services at Sioux Falls, SD, VA Medical Center (GM&S 248 bed hospital). Prefer applicant who is board eligible or certified in internal medicine or family practice. Sioux Falls VAMC is a major teaching affiliate of the University of South Dakota School of Medicine. Academic appointment available to qualified applicants.

Contact: Chief, Medical Service
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Medical & Regional Office Center
P. O. Box 5046
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S Auxiliary News

The South Dakota State Medical Association Auxiliary, for the first time last year, initiated the Sponsor a Spouse program for the benefit of the medical student spouses in our state. Each \$5 donation from an auxiliary member, entitles a medical student spouse to a one year subscription to the National AMA Auxiliary publication, FACETS, and access to the project bank, which is composed of over 900 health related projects, submitted by auxiliaries that have already used the project and it has proved its success, with emphasis in leadership and educational opportunities. This state-wide effort aquaints the student spouses with the auxiliary's many ongoing projects and high ideals, with the hope that these spouses will become volunteer auxilians of the future and establish membership in an active auxiliary.

Last month I had the opportunity to visit with the USD Medical Student Spouses in Vermillion. We talked about the AMA & SDSMA Membership benefits for their spouses as well as auxiliary membership for themselves. During the fun evening, I was very proud to convey to the group that South Dakota physicians support the auxiliary's ongoing AMA-ERF project 100%. These "unrestricted"



funds raised during the year, are presented at the state convention to the Dean of the USD Medical School at the first House of Delegates meeting. With your continued support we hope to better last years figure of \$24,463.47.

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S Future Meetings

April

- Fourth Annual Conference on Critical Care Transport, Palo Alto, CA, April 5-8, Fee: \$195. 18.5 hrs. AMA Category I credits. Contact: Symposia Medicus, 2880 Shadelands Dr., Ste. 404, Walnut Creek, CA 94598. Phone: (415) 935-7889.
- Diabetes, Heart Disease, Cancer, and Pain: Medical & Behavioral Strategies, Coffman Mem. Union, U. of Minn., Minneapolis, MN, April 6-7. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- National Conference on Hospital—Medical Public Policy Issues, Mayflower Hotel, Washington, D.C., April 21-22. Contact: Am. Assoc. for Hospital Planning, 1101 Connecticut Ave., N.W., Ste. 700, Washington, D.C. 20036. Phone: (202) 857-1162.
- Allergy and Clinical Immunology, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, April 21-23. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Current Concepts of Vitreoretinal Disease, Holiday Inn Downtown, Minneapolis, MN, April 25-26. Fee: \$250. 14 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Medical Evaluation for Disability Claims, Marriott Inn, Bloomington, MN, April 29-30. Fee: \$245. 12 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

May

- Family Practice Review: Update 83, Radisson Hotel, St. Paul, MN, May 2-6. Fee: \$375. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Spring Seminar in Medical Imaging, London, England, May 8-11. Fee: \$365. 18 hrs. AMA Category I credits. Contact: Secretary, Spring Seminar, West Park Medical Off. Bldg., 22135 Roscoe Blvd., Ste 104, Canoga Park, CA 91304. Phone: (213) 340-0580.
- Topics and Advances in Pediatrics 83, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 16-17. Fee: \$180. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Congenital Heart Disease, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 23-24. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Current Concepts in Radiation Therapy, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 25-27. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

June

Clinical Hypnosis (Introductory & Advanced) Workshops, Earle Brown Center, U. of Minn., St. Paul, MN, June 10-11. 20 hrs. basic and 8 hrs. advanced AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

- Advances in Gastrointestinal Surgery, Willey Hall, U. of Minn., Minneapolis, MN, June 15-18. Fee: \$400. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- The Sixth Annual Black Hills Seminar on Advances in Clinical Pediatrics, Sylvan Lake Resort, Custer, SD, June 22-24. Contact: Lawrence R. Wellman, M.D., Dept. of Ped., USD School of Med., P.O. Box 5039, Sioux Falls, SD 57117-5039. Phone: (605) 333-7178.
- National Behavioral Pediatrics Conference, Earle Brown Ctr. U. of Minn., St. Paul, MN, June 23-25. Fee: \$300. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

August

1983 Black Hills Summer Seminar, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 11-13. Fee: \$100. 15 hrs. AAFP & AMA Category I credits. Contact: L. H. Amundson, M.D., 3001 S. Holly, Sioux Falls, SD 57105. Phone: (605) 335-5008.

October

The Eighth Annual International Body Imaging Conference, Maui Surf Hotel, Maui, Hawaii, Oct. 8-16. Fee: \$395. 28 hrs. CME Category I credits. Contact: Conf. Secretary, Eighth Ann. International Body Imaging Conf., Dept. of Rad., West Park Hosp., 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (213) 340-0580.

The Sixth Annual Black Hills Seminar

The Sixth Annual Black Hills Seminar on Advances in Clinical Pediatrics—June 22, 23, 24, 1983, at Sylvan Lake Resort, Custer, South Dakota, sponsored by the Department of Pediatrics and Adolescent Medicine, University of South Dakota School of Medicine. Guest faculty include Drs. C. Warren Bierman, Alvin H. Jacobs, Melvin Levine and Philip Sunshine. For complete conference information contact:

Lawrence R. Wellman, M.D.
Program Coordinator
Department of Pediatrics
University of South Dakota
School of Medicine
1100 S. Euclid, P.O. Box 5039
Sioux Falls, South Dakota 57117-5039
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Volume XXXVI April 1983 Number 4

SOUTH DAKOTA JOURNAL OF

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Echovirus 11 Infections in South Dakota, 1979-1980: Spectrum of Disease

Clinicopathological Conference Fifty One Year Old Caucasian Female With Persistent Diarrhea

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A letter outlining the terms of the new participating agreement has been sent to every physician in the state, along with the new participating agreement form.

We encourage you to read the letter and the agreement form as soon as possible. If you have any questions, please contact our office right away because the new participating agreement form must be received in our office by June 1st if you wish to continue your Blue Shield participation.

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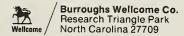
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becise caulion with patients toking more than one of these agents simultaneously USAGE IN PREGNANCY AND LACTA-ION. An Increased risk at cangenital moltarmalians assaciated with minar tranquillizers (meprobamate, chicardiozepoxide, and alozepam) during tirst limester of pregnancy, has been suggested in several studies. Because used transpers, the several studies are accused to the several studies and the several studies. Because used transpers, their use during hits periad should almost always be availed. The possibility that a women at child-bearing patential may be pregnant almost always became pregnant during therapy or inlend to became pregnant from the propositions of the proposition of the proposition

communicate with their physicians about desirability at discantinuing the drug. The drug and passes the placental borrier. It is present both in umbilical-card bload of or near malernal plasma levels and in breast milk at loctating mathers of cancentrations was to four times that of maternal plasma. When use of meprabomate is cantemploted in breastfeeding patients, cansider the drug's higher patients, cansider the drug's higher particular to the drug in the drug is the drug with a point out of reach of children. USAGE IN CHILDREN Keep preparations with aspirin out of reach of children. Equagesic*-Mis not recommended to patients 12 years of gee and under patients 12 years of age and under PRECAUTIONS: ASPIRIN: Sallcylates antagonize uticosuric activity of probene-cid and sulfinpyrazone. Salicytates are reported to enhance hypodycemic ef-fect of sulfonylurea antidiabellas. MEPROBAMATE: Use lowest effective dase, particularly in elderly and/or debi-trated, to proclude over-sedation. Me-probamole is metabolized in the liver and excreted by the kildney; to avoid ex-cess accumulation exercise caution in its use in patients with compromised liver-osionally may precipitate seizures in epi-leptic patients. It should be prescribed cautiously and in small quantities to pa-tients with suicidoi tendencies. ADVERSE REACTIONS. ASPIRIN: May couse epigastra discomment.

cause epigastric discomfort. nauseo, and vomiling. Hypersensitivity reactions, including urlicaria, angioneurotic edema, purpura, asthmo, and anophylaxis may rarely occur. Patients receiving large doses of saltcylates may develop linnitus.

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MEPROBAMATE - CNS: Drowsiness, ataxia, dizziness, slutred speech, head-ache, verifigo, weakness, paresthesias, impoliment of visual accommodation, euphotia, overstimulation, paradoxical exclement, tast EEG activity GI. Nauses, vomiting, diarrhea. CARDIOVASCULAR: Projuitation, tachycatida, various torms of orthythmia, transtent EEG changes, syncope, hypotensive crisis

sient ECG changes, syncope, hypotensive crisis
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established, and thrombocytopenic

purpura. OTHER Exacerbotion of porphyric

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times doily as needed for relief of pain when tension or anxiety is present. Not recom-mended for potients 12 years of age and

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stompot house the removed induction of vomitting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobomate. Aspirin overdosage produces usual symptoms and signs of solicylate inductation. Observation and treatment should include management of hyperthermia, specific patenteral electrolyte therapy for keaccapement of hyperthermia, specific patenteral patenteral patenteral patenteral hyperthermia, specific patenteral hyperthermia, specific patenteral hyperthermia patente OVERDOSAGE: Treatment is essentially

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0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.
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findings of mild-to-moderate symptor of overdosoge, such as stupor or light

of overdosoge, such as stupor or light coma.

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At levels greater than 20 mg percent, more fatalities than survivals can be expected.

Acute combined overdose (meprobamide with other psychotropic drugs or active with other psychotropic drugs of a proposande plus any of these compounds (or of a retaffively low blood of fissue level) cannot be used as a prognostic indicator.

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level) cannot be used as a prognostic indicator. In cases of excessive doses, sleep ensue ropidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiratory assistance. CNS stimulants respiratory assistance. CNS stimulants, seemed courtously as indicated Diuresis, cosmotic (mannital) diuresis, perinand country and proposition of the wrine increases excertion of soil-cylotes. Careful monitoring of urinary output is necessary, and caulton should be laken to avoid overhydration. Relapse and death, after initial recovery, have been attributed to incomplete gastic emptying and delayed absorption. NOW SUPPLIED. Bottles of 50 scored tablets.

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S Medicine

Echovirus 11 Infections in South Dakota, 1979-1980: Spectrum of Disease

Jean F. Kenny, M.D.*

Mary Jo Jaqua-Stewart, Ph.D.†

John F. Barlow, M.D.‡

ABSTRACT

During the warm months of 1979-80, Echovirus 11 infections were common in many areas of the United States with fatal disease occurring in some newborn infants. Echo 11 was isolated from nine patients admitted to Sioux Falls hospitals during the epidemic period. The clinical features of the infections were reviewed. Six of the nine patients were less than two months old and four were less than one week old. In two instances febrile illnesses in mothers at the time of delivery

Echoviruses, along with coxsackie and polioviruses, belong to the large group of enteroviruses, small RNA viruses, that principally infect the enteric tract of man. In temperate climates, infections with these viruses are most prevalent in the summer and early autumn months. During some seasons a variety of enterovirus types are isolated; other years one type predominates and may produce epidemic illness. In the summer and fall of 1979 and early summer of 1980, infections with Echovirus 11 were predominant in many areas of the United States. At that time, Echovirus 11 was also the most fre-

were recorded and in one, probable transplacental infection from an asymptomatic mother occurred. Four patients had aseptic meningitis or meningoencephalitis. Symptoms referable to the central nervous system, respiratory and gastrointestinal tracts were frequent. One baby died but a congenital anomaly unrelated to her infection was the probable cause of death. Patients are discussed in light of previous reports of Echovirus 11 disease.

quent enterovirus isolated in South Dakota. The spectrum of disease produced by this virus varied widely and reports of some cases indicate that disease was severe in some pediatric patients. 4, 5, 6 In this report, case histories of 9 patients admitted to Sioux Falls hospitals from whom Echovirus 11 was isolated are reviewed; clinical aspects of their infections are discussed in light of previous reports of Echovirus 11 disease.

Methods: Throat and rectal swabs or stool collected from patients with suspected viral illness were agitated in Hanks balanced salt solution containing high concentrations of penicillin, kanamycin and polymixin B and transported to the University of South Dakota Clinical Virology Laboratory. Spinal and other body fluids were collected aseptically and transported directly. After clarification by centrifugation or filtration, fluids were inoculated in 0.2 ml amounts in duplicate into three cell lines; primary

7

APRIL 1983

^{*} Department of Pediatrics, East Carolina University School of Medicine, Greenville, NC 27834, formerly from the Department of Pediatrics and Adolescent Medicine, University of South Dakota School of Medicine, Sioux Falls, SD 57105.

[†] Department of Laboratory Medicine, University of South Dakota School of Medicine, Sioux Falls, SD 57105.

Department of Laboratory Medicine, University of South Dakota School of Medicine, Sioux Falls, SD 57105.

Patient	Age	Sex	Diagnosis	Table I Dates of Hospitalization	Features of Illness	Echo 11 Isolate
1	4 days	M	Sepsis Syndrome	8-20-79- 8-25-79	Fever, seizures, lethargy, tachypnea, tachycardia, diarrhea, rash	Stool
2	6 weeks	M	Aseptic Meningitis	8-23-79- 8-27-79	Fever, irritability, rash	CSF
3	32 years	F	Aseptic Meningitis	9-6-79- 9-9-79	Fever, chills, headache, eye pain, pharyngitis, stiff neck	CSF, stool
4	4 days	M	Sepsis Syndrome,	9-15-79- 10-1-79	Fever, diarrhea, hypothermia, rash, jaundice, seizures, lethargy	Stool
5	10 years	F	Nonspecific febrile illness	9-27-79- 9-30-79	Fever, vomiting, headache, chest and abdominal pain	Throat Stool
6	1 day	F	Fetal Hydrops Prematurity	10-6-79	Fetal hydrops, respiratory arrest and death	Ascitic fluid
7	8 weeks	F	Aseptic meningitis, Conjunctivitis	11-7-79- 11-14-79	Seizures, apnea, vomiting, diarrhea, conjunctival discharge	CSF, stoo
8	4 days	F	Necrotizing Enterocolitis, Prematurity	5-25-80- 5-30-80	Bloody stools, Thrombocytopenia	Stool
9	15 years	M	Meningoencephalitis	6-2-80- 6-7-80	Headache, lethargy, vomiting, slurred speech, weakness in extremities	Stool

African green monkey kidney, a heteroploid epithelial cell line (Hep 2) and a diploid fibroblast line, derived from human foreskin (MA 184). Inoculated cells were observed for 2 weeks for the cytopathic effects due to viral replication. If observed, supernatant fluids were passed into additional cell cultures to confirm virus isolation. Blind passages of inoculated, but negative cell cultures were also performed after two weeks and observed for an additional 2 weeks.

Enterovirus positive cultures were forwarded to the State of South Dakota Virology Laboratory where identifications were performed by Elmer Eide, chief technologist, by use of intersecting serum pools.⁷ Hospital charts of patients with positive cultures were reviewed.

Results: Table I summarizes clinical data on nine patients from whom Echo 11 were isolated from one or more sites. Six of the nine patients were less than two months old and four were less than a week old. The only adult was a 32 year old woman who was 2 weeks postpartum. Four of nine patients had aseptic meningitis or meningoencephalitis.

Diagnoses for the five remaining patients were neonatal sepsis syndrome (2), fetal hydrops, necrotizing enterocolitis, and nonspecific febrile illness. Six patients, including those with meningitis, had signs of central nervous system involvement. These were headaches (3), seizures (3), lethargy (4), irritability (2), stiff neck (2), and tense fontanelle (1).

One patient had slurred speech and difficulty moving the upper extremities. Other prominent symptoms and signs included fever lasting 1-7 days in 6 patients (hypothermia alternated with fever in one infant) and gastrointestinal symptoms present in five patients. Gastrointestinal symptoms were diarrhea (4), nausea and/or vomiting (3) and abdominal pain or tenderness (2). Three patients had rashes. In two patients, evanescent macular rashes lasting a day or less and involving mainly the upper portion of the trunk and extremities were described. In one, a maculopapular eruption lasting several days was noted. Respiratory or cardiac abnormalities were observed in 3 patients. One infant and the adult had upper respiratory symptoms, and one infant had episodes of apnea, tachypnea and tachycardia up to 200/min. Two of the older patients described chest pain, myalgia, and/or eye pain. One infant had conjunctivitis and one hyperbilirubinemia which was in excess to what is usually observed physiologically. The family histories of 3 patients revealed that other members of the immediate families had had acute febrile illnesses in the past two weeks. In two instances mothers of newborns who were infected had had febrile illnesses at or just before delivery of their infants. One mother had fever, chills, and chest pain while in labor; the other mother had had fever, nausea and vomiting.

Laboratory studies revealed admission white blood counts in the normal range from 5100/mm³ to

8900/mm³ with 10 to 34% lymphocytes and 55-89% polymorphonuclear cells. Admission spinal fluids on 5 patients with aseptic meningitis contained from 54-154/mm³ cells of which 10-95% were lymphocytes. Three patients had repeat lumbar punctures within 48 to 72 hours of admission. Repeat cerebrospinal fluid cell counts ranged from 50-237/mm³ with 55-100% lymphocytes. Two patients on admission, including one with and one without meningitis, had serum sodium levels less than 130 meq/1 suggesting inappropriate secretion of antidiuretic hormone. With the exception of hyperbilirubinemia in one infant and the positive viral cultures in all patients, other laboratory studies were normal. As noted in table I, Echovirus 11 was isolated from stool in 6, cerebrospinal fluid in 3, throat in 1 and ascitic fluid in 1 patient. In 3 patients virus was isolated from more than one site.

All patients but one recovered. One infant (case #6) born with anasarca and a hypoplastic lung died. Her congenital defect, rather than the infection, was the probable cause of death.

Brief reports of four cases follow:

Case #4

This newborn infant weighed 3274 gm at birth. His mother was a 33 year old Gravida 4, Para 3 whose pregnancy was normal but who became ill with fever, nausea and vomiting at the time of delivery. The baby was vigorous at birth but on the second day of life became febrile to 101°F and had multiple, green, diarrheal stools. Ampicillin and gentamicin were begun. Cultures of blood, urine and spinal fluid for bacteria obtained prior to starting antibiotics were negative. On the third day of life he became lethargic and twitching was noted. A spinal fluid examination showed 13 white blood cells (11 polymorphonuclear), a normal protein and a glucose of 25 mg% with a blood glucose of 35 mg%. He became hypothermic with a temperature of 96.1°F. Jaundice and a pink macular eruption on arms and trunk were noted. The baby required gavage feedings because of lethargy and failure to suck. Fever recurred briefly on the 7th and 8th days of life but after this improvement was rapid and recovery was complete by age 15 days. Echo 11 grew from a diarrheal stool. In addition to his mother, a sibling at home had an acute illness with fever and pharyngitis one week before the baby's birth.

Case #6

This infant was born of a Gravida 3, Para 2 healthy Rh positive mother who was at 30 weeks gestation when she presented to the hospital. Her cervix was fully dilated and fetal membranes were ruptured. The amniotic fluid was meconium stained and the baby presented as a double footling breech.

A grossly hydropic female infant weighing 3109 gms was delivered by Ceasarean section approximately one hour after admission. Apgars were 0 and 1 at 1 and 5 minutes and although the baby made a few gasps, attempts at resuscitation were unsuccessful. At autopsy, there was marked hydrops, with pleural, pericardial, and abdominal effusions amounting to a total of 150 cc of fluid. The left lung was noted to be hypoplastic. Tissues, including the myocardium and placenta were normal histologically. Echo 11 grew from ascitic fluid. There was no history of acute febrile illness in the mother.

Case #7

This 8 week old native American female infant was admitted because of apnea and seizures. The infant weighed 2098 gms at birth and had been hospitalized for 2 weeks in the Neonatal Intensive Care nursery. Subsequently the baby had done well until 10 days before her admission when she developed an upper respiratory infection. One day before admission she had fever to 101°F, mattery eyes, vomiting and loose stools. The day of admission she was noted to have course rhythmic jerking of the extremities, and generalized seizures associated with tonic clonic movements, apnea and cyanosis. Episodes of apnea and bradycardia lasting up to 20 seconds without seizures were also noted. Physical examination was unremarkable but CSF showed 95 white cells/mm³ with 75% polymorphonuclear cells, a protein of 56 mg/dl and normal glucose. Forty eight hours later a second spinal fluid sample, containing 89 cells (63% mononuclear), a protein of 84 mg/dl and glucose of 35 mg/dl. The baby had a serum sodium of 125 meg/1. Gentamicin and ampicillin were begun but were later discontinued when bacterial cultures of blood and spinal fluid proved negative. Gradual improvement occurred with supportive care and the patient was discharged in one week. Echo 11 was isolated from spinal fluid and a stool specimen obtained on admission.

Case #8

This 32 week premature infant was born by Cesarean section because of abruptio placenta. Birth weight was 1550 gms. The baby had respiratory distress. An umbilical catheter was inserted. High oxygen concentrations were administered for 24 hours. At 2 days of age, the baby improved and feedings with breast milk and Enfamil were begun. Moderate hyperbilirubinemia, thought to be physiologic developed. At age 4 days, bloody stools were noted and an abdominal flat plate showed prominent pneumatosis intestinalis involving the colon. A white blood count was 6800/mm³ and platelets were 51,000/mm³. The diagnosis of necrotizing enterocolitis was made. Medical therapy included ampicillin

and gentamicin intravenously, kanamycin orally and fresh plasma and blood. Apnea, bradycardia and increasing bowel distension occurred, however. The patient was taken to surgery where a large piece of necrotic colon was removed and a colostomy performed. A pull-through operation was later accomplished and the patient recovered. Echo 11 was isolated from a diarrheal stool specimen obtained at age 4 days.

Discussion

During 1979 and early 1980, Echo 11 virus was the most common enterovirus isolated in parts of the United States and Canada.³ Forty-four percent of enteroviruses isolated in 1979 and 26% of those isolated during the 1st quarter of 1980 that were reported to the Center for Disease Control were Echo 11.³ This major outbreak occurred in the United States after smaller clusters in 1972 and 1975.² Recent reports suggest that similar Echo 11 outbreaks also occurred in Europe during the late 1970's.^{8, 9, 10} Like our experience, many of the patients with Echo 11 infections who were reported to the Center for Disease Control had aseptic meningitis or encephalitis and the majority of the isolates were recovered from infants and small children.^{2, 3}

Soon after it was isolated for the first time from the intestine of a normal child in 1953, ¹¹ Echovirus 11 has been associated with a variety of syndromes including sporadic and epidemic gastroenteritis, ^{2, 12, 13} upper respiratory disease, ^{14, 15} pneumonia, ¹⁶ myocarditis, ^{2, 17} hepatitis, ^{2, 18} aseptic meningitis and encephalitis, ^{2, 16, 19, 20} paralytic disease, ^{2, 21} nonspecific febrile illness with or without rash, ^{2, 22} and septic illness in the newborn. ^{2, 4, 5, 9, 23} Although aseptic meningitis, with or without encephalitis was the most common clinical entity reported in 1979-80, the illnesses of patients and their contacts were extremely variable suggesting that the strain involved produced heterogeneous illness, with gastrointestinal and respiratory symptoms being particularly common.

A few aspects of our outbreak deserve comment. It is noteworthy that there were 4 newborn infants among our patients. The only adult in the series who was sufficiently sick to require admission to the hospital was a 32 year old woman who had become ill with aseptic meningitis one week postpartum. Although in epidemics, the youngest are most likely to be hospitalized and cultured, some data would suggest that Echo 11 disease may be especially prevalent in the very young.² Reports also imply that women in the peripartum period may be unusually prone to symptomatic infection with this virus. Outbreaks of Echo 11 disease have been reported in maternity units and nurseries in Europe,^{8, 9, 10, 24, 25, 26} Japan,²⁷ Australia,¹⁷ and the United

States. ^{28, 29} Terminally pregnant women, similar to mothers of two of our patients, have had fever, chills, abdominal pain and symptoms of gastroenteritis. On occasion surgery has been performed for possible appendicitis or premature separation of the placenta. ^{4, 6, 8} Most infants who have apparently acquired the infection from their mothers, intrapartum or transplacentally, or from other babies in the nursery or nursery staff, have had poor feeding, irritability, diarrhea and sometimes rash. In some, hepatitis, myocarditis, and an overwhelming sepsis syndrome with shock and disseminated intravascular coagulation have been described. ^{4, 5, 8, 17, 18} At autopsy, prominant adrenal and renal hemorrhages were found. ^{5, 8, 23}

Although stillbirths have been reported with maternal poliovirus infections, their occurrence with other types of enterovirus infections is less documented.³⁰ Recently two separate reports of stillbirths followed Echovirus 11 infections in pregnant women have appeared.31, 32 It is not clear whether the infections were responsible for the fetal loss or incidental to it, although in one case pathologic findings in the infant were similar to those of disseminated enteroviral disease in liveborn infants.³² The role that the Echo 11 virus infection played in the disease and death in the severely hydropic newborn among our patients is also not clear. It seems likely that this infant who survived only minutes after birth and whose ascitic fluid was positive for virus acquired her infection transplacentally. Although peritoneal effusion has been described in some infants with extrauterine Echo 11 infection, 8 it seems likely our baby's abnormal pulmonary anatomy contributed to her massive edema and death. Histologically, tissues did not show evidence of a systemic viral infection.

Also not clear is the importance of the Echo 11 isolate from one infant with necrotizing enterocolitis. Extensive studies attempting to define the etiology of necrotizing enterocolitis have failed so far to incriminate any specific infectious agents as the initiating cause. However, enteroviruses including Echo 11 have sometimes been associated with bloody diarrhea in infants and on occasion have been isolated from infants with necrotizing enterocolitis. All It is likely that infections with enteroviruses, like those with other viruses and bacteria associated with necrotizing entercolitis, are some of a variety of infectious and noninfectious insults to the neonatal intestine which can contribute to the pathogenesis of the disease.

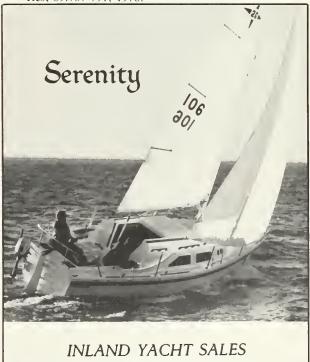
Although Echovirus 11 was isolated from all 9 patients, blood samples for acute and convalescent antibody titers were not available from most patients. Thus, infections could not be confirmed by

rises in serum neutralizing antibodies. This would have been particularly desirable in those patients from whom the virus was isolated only from stool and/or throat. Since serologic confirmation of infection is often not possible or practical, increasingly, clinical virologists have come to accept a naturally occurring enterovirus in the alimentary tract as the probable cause of a syndrome when clinical features are compatible and no other etiology is found.²

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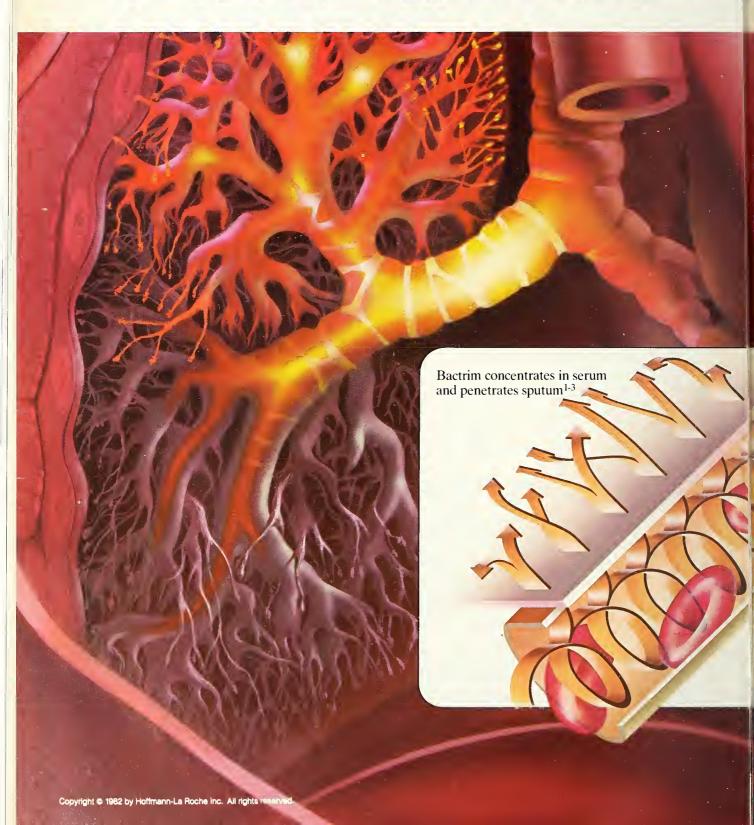
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major pathogens of chronic bronchitis*

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In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens. ^{4,5} One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens. ⁵

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.⁶

Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters. ⁷⁻⁹ Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials

involving nearly 700 patients. **Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

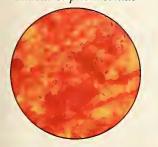
Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

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attacks *H. influenzae*—even ampicillin-resistant strains



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Bactrim

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For ecute otitis medie in children due to susceptible strains of Haemophilus Influenzee or Streptococcus pneumoniee when in physician's judgment it offers an advantege over other entimicroblels. To date, there ere ilmited dete on the sefety of repeeted use of Bectrim in children under two yeers of age. Bactrim ie not indicated for prophylectic or prolonged edminiertetion in otitie media at eny ege.

For ecute execerbetions of chronic bronchitis in adulte due to eueceptible etreine of Heemophilus Influenzee or Streptococcus pneumoniee when in physicien's judgment It offere en edventage over a eingle entimicrobiel egent.

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Contreindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

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PHARYNGITIS. Clinical studies show that patients with group A β -hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopolesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, tever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfa-

Pregnancy: leratogenic Effects: Pregnancy Category C. Because trimetnoprim and suitamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reections: All major reactions to sulfonamides and trimethoprim are included.

even if not reported with Bactrim. *Blood dyscrasias*: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions*: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrotysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions*: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions*: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions*: Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemia agents, sullonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for Infents less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS.

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp (20 ml) b.i.d. for 14 days.

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Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose* packages of 100; Prescription Paks of 20 and 28. Tablets, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 40. Pediatric Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).



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S President's Page



The Annual Meeting of the South Dakota State Medical Association will be held June 2-5, 1983 in Sioux Falls. You know of the Risk Management Seminar Friday afternoon and Saturday morning, which will entitle you to a 10% reduction in your malpractice premium if you are insured by St. Paul Company.

Many of the specialty societies also meet at the Annual Meeting, and I would like to encourage all of you to register at that time.

Concurrent with the Annual Meeting, the South Dakota Political Action Committee (SoDaPAC) will be meeting. This group has been ably led by Ted Wrage for many years. The SoDaPAC Board has recently amended their bylaws to allow for increased representation. These changes were made by them to encourage your participation in political activities.

This is very important to the profession. SoDa-PAC is the cornerstone of our legislative effort. It complements and augments the efforts of our staff when the legislature is in session in Pierre.

A regular membership in SoDaPAC costs only \$40.00 of your personal funds. Sustaining membership is only \$100.00. I ask you to join SoDaPAC and help us with our legislative effort.

This is important to all of us.

Durward M. Laugmo

Durward M. Lang, M.D., President South Dakota State Medical Association

S Auxiliary News



The American Medical Association Education and Research Foundation has added a new Fund in 1983: **The AMA-ERF Medical Student Assistance Fund.** The purpose of the Fund will be to add resources to the student financial aid programs of medical schools, especially the student loan programs at medical schools. The AMA-ERF expects that the Foundation will be able to attract substantial resources for student assistance over the years.

A gift to AMA-ERF that is designated for USD Medical School Loan Fund will be transmitted without a toll exacted for expenses. Thus, a \$100 gift will provide \$100 for our assistance program. The Loan Fund check will be presented at the same time as the unrestricted funds at the annual state meeting in 1984. The South Dakota State Medical Association Auxiliary, with your continued support, will accept the CHALLENGE to provide more dollars for AMA-ERF. We look forward to your participation on June 3, 1983 at the annual AMA-ERF auction held in conjunction with the State Medical Association convention.

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S Clinicopathologic Conference

Fifty One Year Old Caucasian Female With Persistent Diarrhea

David A. Bartnick, M.D.*
Discusser

John F. Barlow, M.D.† Editor

Case #965 554

This 51 year old married caucasian female entered Sioux Valley Hospital with a chief complaint of persistent diarrhea.

Approximately six weeks prior to admission the patient noted a sudden onset of nausea, vomiting and diarrhea. Only the diarrhea persisted and it was unresponsive to kaopectate and diphenoxylate. The patient self prescribed doxycycline without improvement. The diarrhea consisted of loose stools without blood, 6 to 7 times per day unaccompanied by significant abdominal cramping. Her appetite continued as normal but eating did bring on diarrhea. There was a 15 pound weight loss. There were no fever or chills. Two days prior to admission there was swelling of the ankles. Upper gastrointestinal series revealed only some irregularity of the duodenal cap. Intravenous pyelogram and gallbladder x-rays were normal. A chest film was normal. Barium enema showed a redundant sigmoid colon with poor emptying but no other abnormalities. The hemoglobin and erythrocyte sedimentation rate were within normal limits but one of two stools was guaiac positive. 12-panel chemistry examination, electrolytes and carcinoembryonic antigen (CEA) were normal. A 72 hour collection of fecal fat was reported as abnormally elevated.

The remainder of review of systems was unremarkable. The patient had a 45 pack year history of smoking and had had a basal cell carcinoma removed from the nose eight

years previously.

PHYSICAL EXAMINATION: Pulse 96/min. and reg.; respirations 20/min. and reg.; temperature 98.6°F, blood pressure 120 systolic and 80 diastolic. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged and

there were no abnormal sounds or murmurs. Examination of the abdomen revealed no tenderness, spasm, organs or masses. Rectal and pelvic examination were unremarkable. A neurologic examination was unremarkable.

LABORATORY DATA: Urinalysis, light yellow, clear, specific gravity 1.003, negative for protein, glucose, ketone bodies, bile and hemoglobin; sediment 0-2 white cells per hpf. Hemoglobin 14.2 gm/dl, hematocrit 41 vol % with normal red cell indices, white count $7000/\text{mm}^3$ (7 × $10^9/\text{L}$) with a differential of 50% segmented neutrophils, 4% neutrophilic bands, 2% eosinophils, 42% lymphocytes and 2% monocytes. The red cells were normochromic normocytic on smear. A platelet count was $289,000/\text{mm}^3$ ($289 \times 10^9/\text{L}$). Erythrocyte sedimentation rate was 1 mm/hr. Lactic dehydrogenase (LD), alkaline phosphatase, aspartate aminotransferase (AST), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, were within normal limits. Cholesterol was 120 mg/dl. A 5 hydroxyindolacetic acid (5-HIAA) in a 24 hour urine was within normal limits. Serum electrolytes were within normal limits. A d-xylose absorption test revealed plasma levels at one-half hour and one hour of 16 mg/dl and 21 mg/dl (normal 30-40 mg/dl) and a urine level of 0.9 gms in 5 hours (normal 4.1-8.2 gms excreted). A sigmoidoscopic examination was unremarkable. Three stool examinations for ova and parasites were obtained. Stool cultures for salmonella, shigella, Yersinia enterocolitica and Campylobacter jejuni were negative on three occasions. A duodenal aspirate revealed no organisms.

DR. BARTNICK: This case involves a patient with chronic diarrhea of six weeks duration with associated weight loss. Minimal cramping or passage of blood was noted although stools occurred six to seven times daily. Of interest is the fact that ingestion of food seemed to worsen the diarrhea and the fact that various medications gave no improvement.

Because of a multitude of causes leading to diarrhea, the initial history, physical examination, and

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a few general studies need to be evaluated closely to aid in the direction of a more specific workup and diagnosis. The site of the underlying disorder within the bowel is suggested by the character of the stools and the location and quality of any accompanying pain.

One method to help in categorization is distinguishing between a large stool and small stool diarrhea. The "large stool" is often light colored, watery, frothy, greasy, often foul smelling, positive for blood by guaiac, often having undigested food particles and accompanied by intermittent crampy pains in periumbilical or right lower quadrant areas and accompanied by audible borborygmi. These characteristics point to small bowel or proximal colon as the involved site. The "small stool" diarrhea is associated with frequent urges to defecate but passage of only small quantities of feces occurs often only mucus or flatus. Often the stool consistency is mushy or jelly-like with a dark color but is not usually foul smelling. Pain is often gripping, aching, and in lower abdominal quadrants and the sacral region. Passage of flatus, a bowel movement or an enema may result in pain relief. These characteristics point to involvement of lower colon and rectum.

This patient's history describes frequent loose stools without blood which probably describes a ''large stool'' diarrhea although more detailed history would be useful.

Other characteristics noted by history that are helpful would be presence of blood (guaiac positivity) pointing toward an inflammatory, infectious, or neoplastic cause. Pus or exudate would also point toward inflammation or infection. Blood-tinged mucus with lack of odor would lead one to consider shigellosis; a green "pea-soup" quality — salmonella; non-bloody mucus — irritable bowel; mushy, frothy, oily stools — malabsorption; diarrhea that is voluminous and continuous during fasting — a secretory cause; a diarrhea that stops with fasting — an osmotic cause or food allergy; a nocturnal diarrhea — neurogenic sphinctor dysfunction. A perianal fistula or abscess suggests possible Crohn's disease.

A family history of pancreatitis, regional enteritis, ulcerative colitis, multiple endocrine adenomatosis, or of thyroid medullary carcinoma, would also be useful. Possible relationship of exposure to infection or a temporal association with emotional conflicts should also be investigated.

The physical examination of this patient was unremarkable. However, certain findings such as abdominal mass, perianal fistulae or an abscess, anemia, fever, lymphadenopathy, hyperpigmentation, neuropathy, cataracts, lipomas, goiters, ascites, enlarged liver, or rectal masses would certainly give

one an insight as to possible causes of the diarrhea.

Other initial evaluation would include a proctosigmoidoscopic examination with rectal and sigmoid mucosal smears. One would look for ulceration, friability, polyps, and tumor. Certain entities such as ulcerative colitis, amebic proctitis, shigellosis, salmonella and gonococcal proctitis would probably show characteristic changes on inspection. As noted on this patient, the proctosigmoidoscopic examination was negative. Additional studies should include a stool examination for ova and parasites, bacterial culture, examination for Endamoeba histolytica on a warm microscope slide, gram stain for staphylococcal and monilial overgrowth, stool pH (acid pH suggest carbohydrate malabsorption) and phenolphthalien in search of laxative abuse. Other routine evaluations such as complete blood count, 12 panel chemistry survey, and electrolytes, are usually obtained. Any other studies are usually dependent on the individual case. Gastrointestinal x-rays are necessary especially in elderly to rule out organic disease. If an infectious agent is felt to be the cause, blood and urine cultures would be indicated and a barium contrast x-ray would be delayed especially if work-up for possible parasitism was indicated. Recent travel to foreign countries would lead one to consider "travelers diarrhea" with Escherichia coli, Endamoeba histolytica, Giardia lamblia, salmonella, shigella and other agents as possible causes. Usually, diarrhea lasting longer than ten days is probably not "turista" (traveler's diarrhea).

If antibiotic use is in the history, staphylococcal and monilial overgrowth would be sought by examination of stool and mucosal exudate by gram stain. Stool cultures to check for alteration of the mucosal flora and cytotoxicity testing for Clostridium difficile toxin would be indicated. Lincomycin, clindamycin and a variety of other antibiotics may cause diarrhea often due to C. difficile toxin. Neomycin may induce a malabsorption.

If steatorrhea or other evidence of malabsorption are present, studies for d-xylose and vitamin B₁₂ absorption, quantitative stool analysis for fat, abdominal x-ray for pancreatic calcification, small bowel series and biopsy, and small bowel cultures may be indicated.

If neoplasm or inflammatory bowel disease is suspected, barium contrast x-rays would be helpful.

The duration of diarrhea is also very helpful in pointing toward a possible cause. Acute diarrhea (less than two weeks), would be more consistent with a gastroenteritis secondary to viruses, salmonella, food poisoning, poisons, shigellosis, cholera, pseudomembranous colitis (antibiotic use), or travelers diarrhea (enterotoxigenic Escherichia coli). Acute-chronic diarrhea refers to a diarrhea

which begins as an acute diarrhea with or without nausea, vomiting and diarrhea and cramps but continues for a longer period of time. Possibilities include infection by Campylobacter jejuni, Yersinia enterocolitica, Endamoeba histolytica or Giardia lamblia. Campylobacter infection often presents with a colicy, severe abdominal pain with bile and blood-stained diarrhea. Yersinia often gives right lower quadrant pain and x-rays may show signs similar to Crohn's disease. Amebic dysentery is often small volume diarrhea with characteristic changes on proctosigmoidoscopy. Giardiasis has a reservoir in animals both in and outside the US, and is a worldwide infection. Onset is usually within two weeks of arrival in an endemic area but may not occur until months after a trip, etc. Often mild to moderate diarrhea exists and steatorrhea and weight loss may be significant. Small bowel aspirates and/ or biopsy may be necessary to make the diagnosis. Some clinicians feel a trial of atabrine (quinacrine HCL) or metronidazole is warranted prior to an extensive study.

In our present case, stool analysis for fecal fat was abnormally elevated. Stool for ova and parasites was collected but we do not have the results. Three stool cultures were negative for Salmonella, Shigella, Yersinia, and Campylobacter. Because of elevated stool fat, one needs to consider malabsorption as an etiology for the diarrhea. Our case history, in addition, informs us that a d-xylose absorption test was also abnormal and this, again, points to a malabsorption difficulty. A differential diagnosis of the many. entities involved in a malabsorption problem is long and includes many disorders of the pancreas and small intestine. With the rather negative history, physical, laboratory, and x-ray findings, most entities are not likely. Extensive small bowel resection, radiation enteritis, gastrectomy, and druginduced malabsorption are not supported by the history. Intestinal ischemia often gives pain 20-60 minutes following meals and evidence of co-existing atherosclerotic heart disease or brain disease is often present. These factors do not appear to fit our patient well. The various types of sprue usually have characteristic x-ray changes and vitamin deficiencies such as folic acid would present certain characteristic signs or symptoms. Celiac sprue is often suggested by a history of childhood disease and restricting gluten from the diet is highly effective in reversing the disease. Also, there appears to be an association with dermatitis herpetiformis which was not noted in this case. Whipple's disease, amyloidosis or other entities producing malabsorption also usually show changes in x-rays. Whipple's disease often has associated joint involvement, clubbing of the digits, and mental confusion. Intestinal lymphoma is

often noted in people of Jewish and Mexican descent and is hard to differentiate from celiac sprue. Intestinal biopsy would be very useful in further delineating which of these entities is involved. Lymphangiectasia often produces nodular filling defects on x-ray. Food allergies could be a possibility although there is no specific history to suggest this. Eosinophilia was not present. Diabetes mellitus is not suggested by physical examination or laboratory data, nor are scleroderma or any of the endocrinopathies causing diarrhea such as Zollinger-Ellison syndrome (gastrinoma) or Verner Morrison syndrome (watery diarrhea, hypochlorhydria and hypokalemia due to vasoactive intestinal polypeptide).

Parasitism is a good possibility since ova and parasite results are not known and the fact that there are no specific physical findings or history that would rule this out. However, intestinal biopsy would, once again, be very useful in specifying what organisms are involved.

Since the history suggests acute infectious diarrhea followed by a chronic diarrhea with rather mild abnormalities other than malabsorption associated with weight loss, parasitism is a very likely candidate. Because of the nonbloody diarrhea, minimal pain, and minimal findings on proctoscopic and x-ray evaluation, giardiasis would seem to be a very likely candidate. Findings in the stool ova and parasite evaluation, results of small bowel biopsy and possibly a repeat duodenal aspirate would all be helpful in making the proper diagnosis.

Intestinal parasitic infections in man are very common worldwide and becoming more prevalent in the US as foreign travel and immigration increases. Two major groups of parasites are the protozoa and helminths.

The helminths would include tapeworms, roundworms and flatworms. The fish tapeworm, Diphylobothrium latum, is anchored in the small intestine. Infection occurs by ingesting raw fish and is commonly seen in Scandinavia, US, and Canada. Patients often have a vitamin B-12 deficiency — the "tapeworm pernicious anemia." One roundworm (Strongyloidiosis) can produce fatal infection and is found mainly in the rural south. Incidence is very high in Vietnam veterans, immunosuppressed patients and in institutionalized mentally retarded patients. Infectious larvae penetrate the host's skin and then migrate through the venous system to the lungs where they are coughed up, swallowed and then reach the small intestine. Patients may develop steatorrhea, abnormal d-xylose absorption and low serum vitamin B-12 and folate levels. Severe hypoproteinemia due to malnutrition is observed. Capillariasis is found only in the Phillipines. Flatworms such as schistosomes can lead to fibrous, polypoid gastrointestinal lesions, or even stenosis of the small bowel as manifestations of a granulomatous reaction. However, general malabsorption is uncommon.

Protozoan infections include giardiasis, coccidiosis, cryptosporidiosis and malaria. Malaria can give gastrointestinal symptoms of nausea, vomiting, diarrhea and may show abnormal d-xylose, lactose, and vitamin B-12 absorption. However, there is little evidence to support this disease as a cause for general malabsorption. Cryptosporidium is an intracellular protozoan also considered as a coccidium. It can cause severe mucosal lesions of small and large bowel leading to a severe diarrhea but malabsorption is not generally a problem although this is more likely to occur in the immunosuppressed host. Coccidia are intracellular protozoa that are not common pathogens in the US. Often diagnosis by stool examination is difficult even with severe diarrhea and steatorrhea. Malabsorption is felt to be secondary to small intestine mucosal damage.

Giardia lamblia is a flagellated protozoan first described in 1681 by Leuwenhook. It has a world-wide distribution with prevalence varying from 2-50%. In the US, it is both endemic and imported with an overall prevalence of 7.4% in examined stools. Recent reports show the attack rate at 23% for visitors to Leningrad and 36% of servicemen in Vietnam suffered from chronic diarrhea due to Giardia lamblia.

G. lamblia resides in the upper part of the small intestine in man. There are two forms — trophozoite and cyst. The trophozoites are seen in diarrheal stool but not in formed stools, whereas the infective stage (the cyst) is usually found in formed stools. The cyst is quite resistant to chlorination and is transmitted by fecally contaminated food or water and possibly by close personal contact. Diagnosis is usually best made by examining duodenal aspirate if the stools are negative.

There appears to be an increased incidence of giardiasis in children, patients with hypochlorhydria or achlorhydria and in certain immune deficiency states including acquired dysgammaglobulinemia and X-linked hypogammaglobulinemia.

Usually most adults are asymptomatic but infection can lead to acute gastrointestinal distress as well as chronic diarrhea and malabsorption and may even be severe enough to be mistaken for a case of celiac sprue. Standard tests such as fecal fat and nitrogen, d-xylose absorption, serum folate and carotene are frequently abnormal. Vitamin B-12 levels may also be low.

X-rays of the small bowel can show thickening and distortion of the mucosal folds. Small intestinal biopsies range from normal to that of complete villous atrophy. Other factors beside direct invasion which may be involved in the pathogenesis of malabsorption in giardiasis include a direct mechanical barrier to absorption, competition for nutrients directly, and altered intestinal motility. Bacterial overgrowth may accompany giardiasis and may also be partly responsible for associated steatorrhea. High luminal free bile acids caused by bile salt deconjugation may also have a role.

Successful treatment with quinacrine or metronidazole has usually cured the malabsorption. Quinacrine (Atabrine) is given as 100 gm. three times a day for seven days. Metronidazole (Flagyl) is given as 250 mg. three times a day for 7-10 days. Higher doses for several weeks may be needed in the immunologically deficient patient. If the steatorrhea continues even though the parasite appears to have been eradicated, broad spectrum antibiotics have been of benefit. It should be noted that lactose deficiency and other enzyme deficiencies may persist in the small bowel after giardiasis. Cramps and diarrhea after ingestion of milk may simulate persistent infection.

Dr. Bartnick's Diagnosis Giardiasis

DR. BARLOW: As Dr. Bartnick surmised, the three stool examinations for ova and parasites showed many Giardia lamblia cysts. Because of a negative duodenal aspirate and because there was severe malabsorption, a small bowel biopsy was done. Focally, there was some flattening of the normal villous architecture as well as a chronic inflammatory infiltrate in the lamina propria. The striking



Figure 1
Giardia lamblia trophozoites with characteristic shape in intervillous space of small intestine.

finding, however, was in the lumen of the bowel where many organisms consistent with trophozoites of Giardia lamblia were seen (Fig. 1).

FINAL ANATOMIC DIAGNOSIS GIARDIASIS

DR. JOHN D. BARKER, JR.*: That was a nice discussion. Giardisis is not uncommon. It is indigenous to South Dakota. This patient had never left the state for the more popular mountain regions where giardiasis is known to be endemic. In Colorado they call it Beaver Fever. Visitors to these mountainous areas often drink from the clear mountain streams. not realizing that a beaver upstream has polluted the water with viable giardia cysts. Other animals can also harbor this organism. Numerous outbreaks have occurred. The outbreak in Leningrad was the most famous. There was a large outbreak in Rome, New York from a contaminated city water supply. As has been pointed out, the pathogenesis of the malabsorption in giardiasis is largely unknown. I have found that stool examination has usually indicated the presence of the organisms without having to resort to a duodenal aspirate. A purged stool using magnesium citrate is often necessary to find the organism in the stool. My choice of therapy is metronidazole. This may have to be given for several courses. If diarrhea persists, one must either look for other causes or check to see whether reinfection has occurred.

DR. RANDE SHORT†: Should you treat family members?

DR. BARKER: Only if they are symptomatic.

DR. BARLOW: I discussed this case because we could show that the organism can be demonstrated on small bowel biopsy. It can also be demonstrated very nicely by duodenal aspirate and I cannot explain why that procedure did not demonstrate the organism in this case. More common is the reverse situation in which Giardia lamblia can be recovered on duodenal aspirate but not in the stool. There is a serologic test for this disease but it is not commercially available at this time. However, specific serum antibodies are demonstrable in about 90% of patients who have had giardiasis. I should mention that giardiasis can produce acute, subacute or chronic diarrhea. The latter two are characterized by malabsorption. The patient may suffer any one of these manifestations independently or can develop chronic disease after an acute episode of giardiasis.

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Chapter News

Refor the 80s





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National CME Survey

Nearly one thousand physicians responded to a recent CME Survey conducted by the CME Bulletin, published by James E. Sinkinson.

While these physicians singled out numerous aspects of CME in need of a cure, they also offered sound recommendations as to how CME providers can improve service and increase attendance. Clearly, however, doctors' biggest complaints centered on the high costs associated with CME, an indication that the recession's effects are now being felt measurably among medical professionals.

Question: Do you think primary care physicians should be required to maintain competence through mandatory CME?

Yes 72% No 25% No answer 3%

Question: What are your three biggest complaints about CME conferences you've attended?

High cost	73%
Poor speakers	34%
Poorly prepared written materials	29%
Information not geared to needs	23%
Poorly organized	19.5%
Poor scheduling	15%
No complaints	10%
Lack of family activities	6%
Information not up to date	5.5%
Too much leisure time	4%
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Question: Which of the following CME activities will you use to meet your Category 1/Prescribed CME requirements?

Regional seminars	79%
Self-study programs	61%
National conferences	56%
Association journals	46%
Other	19%

Question: What is your first preference for a conference location?

Close to city 1 live in	48%
Major US city	22%
Luxury resort	14%
Outdoor sport location	13%
Hospital or university	9%
No preference	6%
Foreign country	5%
Cruise ship	4%
-	

Question: Which conference formats do you find most useful?

Seminars to small groups (25 people)	54%
Lectures to large groups (100-200 people)	38%
Laboratory workshops	12.5%
Round-table discussions	7.5%

Question: What is the best time of year for you to attend a CME conference?

March-April	30%

January-February	23%
September-October	22%
July-August	17.5%
May-June May-June	16%
November-December	10%
No preference	3%

Question: Do you plan to take vacation with your CME activities?

Question: Which four CME topics are of greatest interest to you for your personal CME needs?

you lot your personal City	
Topic	AAFP
Cardiovascular disorders	55%
Geriatrics	31.5%
Dermatologic disorders	31%
Gynecological disorders	29.5%
Abdominal and GI disorders	29%
Musculoskeletal disorders	27%
Infancy and childhood disorders	22%
Manipulative procedural skills	20%
Hormonal metabolic and	
nutritional disorders	18%
Sports medicine	16.5%
Thoracic/respiratory disorders	15%
Psychologic and psychiatric disorders	14%
Practice management	13%
Urogenital disorders	9%
Nervous system disorders	9%
Disorders due to chemical and	
physical agents	8%
Practice marketing and PR	6%
Hematopoetic disorders	5%
Head and neck disorders	3%

CME and the Future

These survey results are certain to stir conversation among physicians and CME providers alike. If conference organizers were not already convinced of the fact, these physician reactions should go far to convince them that the Eighties will be what one doctor called "a Darwinian period." Not only are providers struggling for survival in one of the most difficult economic times in decades, but they face increasing cynicism from their constituency. The survival of many CME programs will no doubt depend on more astute and creative approaches by organizers — and more attention to the actual needs of the physicians asked to attend their activities.

Medical Missionary to Speak

Maynard Seaman, M.D., South Dakota native and 1954 graduate of USDSM, will be the SDAFP noon luncheon speaker at the forthcoming SDSMA convention in Sioux Falls.

Dr. Seaman will speak on "The Mycobacteria of Asia" at the 12:45 p.m. luncheon on Saturday, June 4, at the Ramada Inn.

Plan to attend.

S Future Meetings

May

- Topics and Advances in Pediatrics 83, Mayo Mem. Aud., U. of Minn., Minneapolis MN, May 16-17. Fee: \$180. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Congenital Heart Disease, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 23-24. AMA Category I credits. Contaet: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone (612) 373-8012.
- Current Concepts in Radiation Therapy, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 25-27. AMA Category I credits. Contaet: CME, Box 293 Mayo Mem. Bldg., 420 Delawarc St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

June

- Bariatric Surgery Colloquim, U. of Iowa, Iowa City, IA, June 2-3. AMA Category 1 credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.
- Legal and Ethical Aspects of Health Care For the Elderly, Mayflower Hotel, Washington, D.C., June 2-4. Fee: \$250. Contact: Am. Society of Law & Med., 765 Commonwealth Ave., Boston, MA 02215.
- Recent Developments in Mental Health Law, Ambassador West Hotel, Chicago, IL, June 9-10. Fee: \$200. 12 hrs. AMA Category I credits. Contact: Am.Soe. of Law & Med., 765 Commonwealth Ave., 16th Fl., Boston, MA 02215. Phone: (617) 262-4990.
- Advanced Cardiac Life Support, Waterloo, IA, June 10-12. AMA Category I credits. Contact: Richard Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.
- Advanced Cardiac Life Support, Spirit Lake, IA, June 14-16. AMA Category I credits. Contact: Richard Caplan, M.D., Assoe. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.
- The Sixth Annual Black Hills Seminar on Advances in Clinical Pediatrics, Sylvan Lake Resort, Custer, SD, June 22-24. Contact: Lawrence R. Wellman, M.D., Dept. of Ped., USD Sehool of Mcd., P.O. Box 5039, Sioux Falls, SD 57117-5039. Phone: (605) 333-7178.
- National Behavioral Pediatrics Conference, Earle Brown Ctr. U. of Minn., St. Paul, MN, June 23-25. Fee: \$300. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Advances in Gastrointestinal Surgery, Willey Hall, U. of

Minn., Minneapolis, MN, June 15-18. Fee: \$400. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

July

Medical Problems of Musicians Conference, Aspen Musical Festival, Aspen, CO, July 27-Aug. 1. 20 hrs. AMA Category I credits. Contact: Aspen Music Festival, 1860 Broadway, #401, New York, NY 10023. Phone: (212) 58I-2196.

August

- 1983 Black Hills Summer Seminar, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 11-13. Fee: \$100. 15 hrs. AAFP & AMA Category I credits. Contact: L. H. Amundson, M.D., 3001 S. Holly, Sioux Falls, SD 57105. Phone: (605) 335-5008.
- Annual Sixth District American College of Obstetricians and Gynecologists Meeting, Rushmore Plaza Civic Ctr., Rapid City, SD, Aug. 18-20. Contact: The Women's Clinic, 2805 Fifth St., Ste. #110, Rapid City, SD 57701. Phone: (605) 343-6550.
- Controversies and Consensus in Obstetrics, Greek Island Cruise, Aug. 2I-Sept. 5. Fee: \$295. AMA Category I credits. Contact: Symposia Medicus, 2880 Shadelands Dr., Ste. #404, Walnut Creek, CA 94598. Phone: (415) 935-7889

September

Atlantic-Mediterranean Meeting on Gastroenterology, Mediterranean Cruise, Sept. 17-Oet. 7. Fee: \$1,640 includes passage, meals, and other extras. Contact: Mario Blanco Peres, Rua Goncalo, Cristovao, 116-3°, 4000 Porto, Portugal. Phone: 24294, 20933 or 31002. Telex: 26850 FRZDN-P.

October

The Eighth Annual International Body Imaging Conference, Maui Surf Hotel, Maui, Hawaii, Oct. 8-16. Fee: \$395. 28 hrs. CME Category I credits. Contact: Conf. Secretary, Eighth Ann. International Body Imaging Conf., Dept. of Rad., West Park Hosp., 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (213) 340-0580.

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As a cost containment effort, South Dakota Blue Shield will be cancelling all current participating agreements as of June 30, 1983, and will issue new ones, which if signed by the physician, will become effective July 1, 1983.

A letter outlining the terms of the new participating agreement has been sent to every physician in the state, along with the new participating agreement form.

We encourage you to read the letter and the agreement form as soon as possible. If you have any questions, please contact our office right away because the new participating agreement form must be received in our office by June 1st if you wish to continue your Blue Shield participation.

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Clinicopathological Conference Sixty-One Year Old Caucasian Man With Rheumatoid Arthritis and Renal Failure

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Clinicopathologic Conference

Twenty-Two Year Old Caucasian Male With Persistent Hypertension

Richard P. Day, M.D.*

Barry Lankhorst, M.D.;

John Sall, M.D.‡

Discussers

John F. Barlow, M.D.§ Editor

Case # 958 797

This 22 year old caucasian male entered Sioux Valley Hospital with a chief complaint of high blood pressure. Diastolic pressures up to 110 mm of Hg and systolic pressures over 160 mm of Hg had been recorded.

The patient four years previously was noted to have high blood pressure without previous history during a routine physical examination for military service. Since that time, the blood pressure had increased and was partly controlled on conventional medication. Laboratory evaluation at another hospital revealed a normal hemogram and 12 panel chemistry including blood urea nitrogen and serum creatinine. An intravenous pyelogram (IVP) revealed a 10 cm. cyst in the right kidney. It was found that the patient had been hit sharply in the back during a football game six years prior to admission and may have developed an episode of hematuria. The history is not clear on that point. He had also suffered a fractured clavicle. The patient had had extensive evaluation at outlying facilities for hypertension. At present the hypertension was under fair control with minoxidal, hydrochlorthiozide and atenolol.

The father had mild hypertension easily controlled with medication. There was no other history of hypertension in the family. One grandfather had died of myocardial infarction at age 69, another at 78 of the same disease.

PHYSICAL EXAMINATION: Well developed, well nourished caucasian male, blood pressure 140 systolic and 90 diastolic; pulse 80/min and regular; respirations 20/min and regular; temperature, afebrile. Examination of the head and neck was unremarkable except for Grade I constrictive changes in the fundal vessels. Significant sclerosis, hemorrhages, exudate or papilledema were not present. The carotid pulsations were 4+. The lungs were clear to auscultation and percussion. There was a faint, short bruit over the left clavicle, which had previously been fractured. This was not heard by all observers. The heart had a normal rhythm and was not enlarged. There were no murmurs or gallop rhythm. Examination of the abdomen revealed no palpable organs, masses, spasm or tenderness. No abdominal or flank bruits were noted and genital examination was unremarkable. The extremity pulses were 4 + and equal. There was no delay in the femoral compared to radial pulses. The neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis, light yellow, clear; specific gravity 1.007, pH 8.0; negative for protein, glucose, ketone bodies, bile and hemoglobin; sediment — negative; hemoglobin 15.3 gm/dl; hematocrit 46 vol/dl; with normal red cell indices. Total leukocyte count 7,500/mm3 (7.5 x 10°L) with 78% neutrophils; 2% neutrophilic bands; 3% eosinophils; 16% lymphocytes and 1% monocytes. The platelet count was 202,000/mm3 (202 x 10°L). The red cells were normochromic, normocytic on smear. Sodium 139 meq/L, potassium 4.4 meq/L, chloride 105 meq/L, CO2 25 meq/L, blood urea nitrogen 15 mg/dl; creatinine 1.3 mg/dl. The remainder of the 12 panel chemistry was unremarkable. An electrocardiogram was within normal limits. Twentyfour hour urine determination of catecholamine metabolic products (VMA), metanephrines, and free catacholamines were within normal limits. A renal angiogram revealed no evidence of renal artery stenosis in the primary or secondary

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branches. There was no evidence of aneurysm. There was an abnormality at the lower pole of the kidney but no evidence of tumor vessel formation. A computer tomographic scan (CT) of the kidneys showed a cystic mass at the right lower pole of the kidney. Ultrasound examination confirmed this finding. Needle aspiration of the mass under ultrasound direction showed no malignant cells. Renal vein renin determinations before furosemide showed right renal vein 0.7 ng/ml/hr, left renal vein 0.6 and inferior vena cava 0.6 and after furosemide right renal vein 2.4, vena cava above renal 2.0, left renal vein 0.9. The renin determinations were interpreted as absence of definite evidence of unilateral renal disease. A surgical procedure was performed.

DR. DAY: A patient may be called hypertensive if, at least on three occasions, arbitrary limits of a systolic pressure of 140 mm the Hg and diastolic pressure of 90 mm of Hg are surpassed. It is estimated that 25% of American adults have diastolic pressures of greater than or equal to 90 mm of Hg.

In the initial approach to an individual with elevated blood pressure, a physician needs to determine whether the person has fixed or labile hypertension. Approximately 50% of the patients will have labile hypertension. These patients may benefit from stress reduction techniques and still need to be followed up at least yearly since they may have an increased risk of becoming hypertensive later in life. The exceptions to this "wait and see" approach include evidence of accelerated hypertension (a diastolic pressure of greater than or equal to 125 mm of Hg) or signs of target organ damage. In this case, hospital workup is indicated.

A realization that 90% of cases of hypertension is essential may be a comment on our lack of understanding of the disease. Of the remaining 10%, 5% will have surgically curable disease.

Suggested factors in the increased likelihood of surgical cure are:

- 1. When onset of hypertension occurs at 25 years of age or younger.
- 2. Hypertension that is poorly controlled by medication.
- 3. Malignant hypertension.
- 4. Sudden acceleration of a previously well controlled hypertension.
- 5. Onset of hypertension after 60 years of age.
- 6. An abdominal bruit.

Every hypertensive individual deserves a history and physical evaluation. Previous history of cardiovascular disease or renal disease should be noted. Family history of cardiovascular disease, hypertension, or renal disease should also be elicited. Other important information relative to the differential diagnosis include any medications, type of diet and any history of flank trauma.

Physical examination should include at least standing and recumbent blood pressures, in both the

left and right arms, as well as blood pressures in the lower extremities, especially in young individuals, in search of coarctation of the aorta. The radial and femoral pulses should also be compared to rule out any obstructive vascular disease. Ocular fundi should be examined for retinopathy; cardiac and neurologic status should be evaluated; and the abdomen should be examined for any masses or abdominal bruits.

Laboratory evaluation is really dependent on the history and the physical signs that are elicited. Routinely done laboratory tests include: urinalysis, hemoglobin, blood urea nitrogen, creatinine, uric acid, blood sugar, electrolytes including HCO3, cholesterol, triglycerides, thyroxin (T4), and an electrocardiogram. Common tests for specific entities include IVP, CT and ultrasonography of the abdomen, arteriogram or digital subtraction angiography to evaluate kidneys and renal vasculature, renal vein renin assays to determine if unilateral renal disease is present, plasma or urinary steroid determinations to indicate Cushings disease or hyperaldosteronism and plasma or urinary catecholamine studies for pheochromocytoma. Saralasin testing may also be used as indication for hyperreninemia. It is a competitive inhibitor of angiotensin II. The most common causes of hypertension in a young individual are as follows:

- 1. central hypertension 89%
- 2. chronic renal disease 5%
- 3. renal vascular disease 4%
- 4. coarctation 1%
- 5. primary hyperaldosteronism 0.5%
- 6. pheochromocytoma or Cushing's 0.2%

Primary hyperaldosteronism generally occurs in a young, mildly hypertensive female. These patients have symptoms of polyuria and muscular weakness and almost always have hypokalemia (less than 3.0 meq/L) which is often detected on electrocardiogram and can lead to cardiac arrhythmias. Seventy-five percent of cases are caused by an adrenal adenoma and the remaining 25% by micronodular hyperplasia. The triad of hypertension, hypokalemia and low plasma renin levels are a classic triad for primary hyperaldosteronism. The confirmatory test is measuring urinary aldosterone. Further tests need to be conducted to differentiate between the primary types.

Pheochromocytoma is rare and expected clues of headache, fever, tachycardia and periods of extremely elevated blood pressure occur only in one-third of the cases. Many investigators recommend that screening of urinary catecholamines be undertaken in any young hypertensive. Ninety percent of the tumors are located in the adrenal medulla. At

least 10% appear to be familial and any documented catecholamine excess deserves family screening.

Cushing's syndrome is usually recognized by its physical characteristics. There is a classic moon facies, the buffalo hump and truncal obesity with abdominal stria. Hypertension is almost always present in these individuals. The diagnosis of Cushing's syndrome depends on the demonstration of an increased cortisol production.

Coarctation of the aorta can be suspected after physical examination. A cardiac murmur, a delay of femoral compared to radial pulses, blood pressures that are markedly decreased in the lower extremities, as compared to the upper extremities, and a general body habitus with a muscular large thorax in comparison to small, underdeveloped lower extremities are good clues for this diagnosis. Chest x-ray may also show the classic rib notching secondary to the dilated intercostal arteries from the increased collateral flow. A figure 3 sign, which is due to indentation of the ascending aorta, may also be seen on chest x-ray. Abdominal coarctation is rare and an abdominal bruit may not be heard, but the physical findings are similar to coarctation of the ascending thoracic aorta.

In thyroid and parathyroid disease, hypertension is often a secondary problem. Hypertension in hyperparathyroidism is believed to be due to the hypercalcemia and is reversed by treatment of the underlying disease. The etiology of hypertension in myxedema is unknown but it resolves after treatment with thyroid hormone. Approximately one-third of hyperthyroid individuals are hypertensive. This is probably from the increased sympathicomimetic activity. Treatment of the disease process also reduces the blood pressure.

Drugs involved in elevating blood pressure are estrogens, amphetamines, sympathicomimetics, monamine oxidase inhibitors and licorice that contains glycyrrhizinic acid which has a mineralocorticoid effect.

Neurogenic hypertension is associated with increased intracranial pressure in the central nervous system such as a subdural hematoma. Reye's syndrome or any of the encephalitides can also cause elevated blood pressures due to increased intracranial pressure. Peripheral neuropathies as in porphyria, lead poisoning or tabes dorsalis may be associated with transient hypertensive episodes.

Renal and renovascular diseases are the most likely etiology of secondary hypertension. This may be bilateral or unilateral. Renal vascular hypertension would be suggested by refractory and progressive hypertension before the age of 20. Abdominal bruit is present 50% of the time with renal artery stenosis. Unilateral disease is potentially remediable.

Findings on intravenous pyelography (IVP) include:

- 1. slow uptake of the contrast media unilaterally.
- 2. significant discrepancy in renal size.
- 3. late hyperconcentration of a contrast media on one side.
- 4. scalloping of ureters, which suggests the presence of ureteral pressure from collateral vessels.
- 5. delayed washout with diuresis.

IVP can fail to document existing disease 25% of the time. Unfortunately, positive findings occur with essential hypertension 11% of the time. This makes further evaluation necessary.

Angiography is a test of choice for demonstrating vascular pathology and renal vein renin assays for demonstrating unilateral renal disease.

Angiographic findings in significant renal artery stenosis due to fibromuscular hyperplasia or atherosclerosis include:

- 1. stenosis greater than 70% of the luminal diameter
- 2. collateral circulation.
- 3. poststenotic dilatation.
- 4. diminished velocity of flow across the stenosis.
- 5. pressure decreases of greater than or equal to 25% of the systolic pressure across the stenosis.

In the case presented today, there is no evidence of any renal vascular disease by angiography.

Renal parenchymal diseases are therefore in the top line for consideration. Negative findings on the history and physical, urinalysis, blood urea nitrogen and creatinine eliminate such diseases as glomerulonephritis, acute and chronic pyelonephritis, diabetic nephropathy, interstitial nephritis and connective tissue disease. Many other entities were clearly not present on IVP, ultrasound or CT such as polycystic kidney disease and hypernephrosis.

In this young individual, the findings are a perirenal mass with cystic changes which are confirmed by IVP, ultrasound, angiography and CT. In this case, trauma to the flank is a very important clue. The angiographic data showing an avascular mass leads us away from the diagnosis of a variety of renin hypersecreting tumors and toward a diagnosis of Page kidney secondary to renal trauma. Page demonstrated in 1939 that a dog's kidney wrapped in cellophane developed subsequent hypertension. In 1955 a young adult was found to have a subcapsular perirenal hematoma presumed to cause renal ischemia by compression and hypertension secondary to

stimulation of the renin — angiotension system.

The traditional question of flank trauma is a good leading clue. Recent trauma with a subcapsular hematoma should be followed because, although most resolve spontaneously, a minority will lead to the classic perirenal fibrosis necessary for the Page kidney to develop.

There are other mechanisms leading to the development of the Page kidney besides trauma. These include renal surgery, renal biopsy, infection, arterial disease and blood disorder.

Diagnosis of this entity has rested in the past with IVP and renal angiography, but sonography and CT are becoming increasingly important modalities. There is enlargement of the kidney but the pyelocayceal system is often unaffected. There can be various degrees of decreased function. One study described an avascular mass causing a concavity of the adjacent parenchyma. Ultrasound has demonstrated solid masses with cystic changes or perirenal fluid collection. CT is valuable because of its ability to demonstrate different densities, as well as function.

Angiography is still one of the most valuable studies but it is also invasive. It is essential in most cases because these masses may be tumor and the surgeon needs to know the preoperative vascular supply. In one case, where no trauma was demonstrated, an abnormal dual vascular supply was found and thought to be the cause of the hypertension. Generally Page kidney is associated with an avascular perirenal process with compression of adjacent parenchyma. Segmental and interlobar arteries are stretched and attenuated and there may be cortical margin irregularity.

Gross morphology usually demonstrates a thick, fibrous capsule with multiple small perirenal cysts in fatty tissue and an underlying contracted kidney. Microscopic examination shows chronic inflammatory changes with fibrosis of the capsule. There may be hyperplasia of the juxtaglomerular apparatus or hyalinization of the glomeruli.

Corrective surgery can be as simple as decortication of a dense capsule or evacuation of the hematoma, but this is an exception rather than the rule. The duration of the lesion is an important factor in the determination of the post surgical results. In many instances only nephrectomy may improve the hypertension.

I left discussion of the renin determination until last because, in general, the renin values should show a lateralization in the Page kidney. In this case, no definite lateralization by any criteria was noted.

Renin was discovered in 1898 and although it was thought to be related to hypertension, it was also known, at that time, that it had no vasopressive action. Renin is predominantly synthesized in the kidney, but it has been shown to be a major renin source. The juxtaglomerular apparatus between the afferent and efferent arterioles in the glomerulus is the site responsible for renin release. In periods of decreased renal perfusion, renin is increased.

Renin hydrolyzes angiotensinogen (from the liver) at the leucine bond to produce a decapeptide angiotensin I. Angiotensin I is a substrate for angiotensin converting enzyme present in pulmonary vascular endothelium. In a single pass through the lungs, angiotensin I is changed from angiotensin I to angiotensin II which is one of the most potent vasoconstrictors known (about 50 times the vasoconstricting power of norepinephrine) and is a mediator for aldosterone secretion which is in turn responsible for sodium reabsorption and potassium excretion.

If one used a renin ratio of 1.5:1 between the two kidneys to predict a surgical cure, there is a 2% false positive and a 20% false negative rate. The Vaugham scoring system uses as its criterion on a peripheral renin assay an index calculated on the bases of a 24 hour urine sodium and the renin levels from the ipsilateral and contralateral kidney. This lowers the false positive rate to 0% but the false negative rate increases to 33%.

There are problems that are inherent in the testing of renal vein renin assays. One is the replacement of the catheter. Also any variation in anatomy may change the results. Diet or drugs affecting renin secretions and body posture while taking the test all have significant effects. Just a change in the laboratories performing the tests may radically alter the interpretation of the renin results.

There are documented cases of reverse ratios of 1.9 and 3.4 where there was actually less renin secreted from the kidney with renal artery stenosis. This variation is due to both a reduction in renal blood flow and shrinkage of the affected kidney.

Hopefully converting enzyme inhibitors such as captopril in addition to furosemide can be used with renal renin vein assays to accentuate the differences in the renal vein renin values and make this a more definitive test.

Dr. Day's Diagnosis Unilateral Renal Disease ? Page Kidney

DR. BARLOW: I would call your attention to the arteriogram and computer CT. The former reveals an avascular lower pole of the right kidney with no evidence of tumor blood vessels (Figure 1). The computer tomogram shows cyst formation at the periphery of the right kidney (Figure 2).

DR. LANKHORST: I would like to give credit to the

medical student who obtained the history of flank trauma.

One would usually expect the renin determinations to show appropriate inequality if indeed this represents unilateral renal disease producing hypertension. However, this patient had hypertension for six years and probably nephrosclerosis secondary to the hypertension may have been produced in the contralateral kidney over a period of time. This phenomenon of secondary nephrosclerosis in the contralateral kidney often explains equalization of renin values even though unilateral renal disease is the original culprit. I would estimate that such a situation occurs in 10-15% of cases in which unilateral renal disease is the cause of hypertension.

Another point is that renin values are notoriously



Figure 1
Renal Angiogram showing avascular and distorted right lower pole of kidney (viewer's left) arrow.

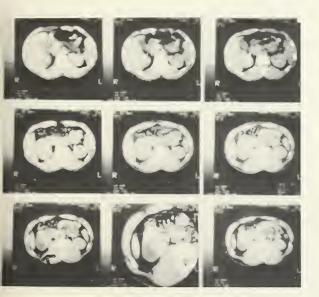


Figure 2
Computer tomogram showing kidney on right (viewer's left) enlarged and cystic compared to left (arrow).

difficult to perform in a reproducible manner even in laboratories which run these tests daily. If a patient has other studies which suggest hypertension caused by unilateral renal disease, you must often ignore the lack of lateralization of renin values.

DR. SALL: I approached the kidney through a transabdominal incision. The surface of the right kidney was hard and irregular. I was concerned about neoplasm and performed a radical nephrectomy.

The patient became hypotensive three hours postoperatively but this responded to volume expansion. The patient is now normotensive without medication (three months after surgery).

DR. BARLOW: We received the entire right kidney with surrounding fat. There was a hard irregular fibrous surface. On cross-section much of the kidney was encased in a white hard thickened fibrous rind. At the lower pole there was a cavity in the rind filled with grumous brown material (Figure 3). Microscopic examinations revealed extensive fibrosis. The cavity described was an organizing hematoma. I feel that the densely adherent fibrous process could not have been removed by decortication. The gross picture is not dissimilar to a case reported by Grim et al in the **Journal of the American Medical Association** in 1975. ¹ It is reasonable to relate the process to the previous trauma and indict the encapsulation of the kidney as a cause of hypertension.



Figure 3
Thickened rind of fibrous tissue partially encircling kidney.

FINAL ANATOMIC DIAGNOSIS FIBROUS ENCAPSULATION OF RIGHT KIDNEY SECONDARY TO TRAUMA (PAGE KIDNEY)

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Brief Prescribing Information

CONTRAINDICATIONS: Patients with severe hypertension, severe coronary artery disease, and in patients on MAD inhibitor therapy, narrow-angle glaucoma, urinary retention, peptic ulcer, during an asthmatic attack.

asthmatic attack. Hypersensitivity: Contraindicated in patients with hypersensitivity or diosyncrasy to sympathomimetic amines or phenanthrene derivatives. Nursing Mothers: Contraindi-cated because of the higher than usual risk for infants from sym-pathomimetic amines.

cated because of the higher than usual risk for infants from sympathomimetic amines.

MARNINGS: Use judiciously and sparingly in patients with hypertension, diabetes mellitus, ischemic heart disease, increased intraocular pressure, hyperthyroidism, or prostatic hypertorphy. May produce CNS stimulation and convulsions or cardiovascular collapse with accompanying hypotension.

Use with caution in patients with increased intraocular pressure, cardiovascular disease, hypertension or in patients with a history of bronchial asthma. Do not exceed recommended dose.

Use in Elderly: The elderly (60 years and older) are more likely to have adverse reactions to sympathomimetics. Dverdosage in this age group may cause hallucinations, con-

group may cause hallucinations, con-vulsions, CNS depression and death. PRECAUTIONS: General: Should be

used with caution in patients with diabetes, hypertension, cardiovascular disease and hypertensicular disease and hypertensicity to ephedrine. The antihistaminic may cause drowsiness and ambulatory patients who operate machinery or motor vehicles should be cautioned accordingly.

accordingly.

Information for Patients: Antihisimbornation for Patients: Antium and physical abilities required for the performance of potentially hazard-ous tasks, such as driving a vehicle or operating machinery, and mental alertness in children.

Dug Interactions: MAD inhibitors and heat addresses in children.

brug interactions: May inno-itors and beta adrenergic blockers increase the effect of sympatho-mimetics. Sympathomimetics may reduce the antihypertensive effects of methyldopa, mecamylamine, re-serpine and veratrum alkaloids.

of methyloopa, mecamylamine, reserpine and veratrum alkaloids.
Concomitant use of antihistamines
with alcohol, fricyclic antidepressants, barbiturates and other CNS
depressants may have an additive
effect.

Pregnancy Calegory C: Animal
reproduction studies have not been
conducted with NOVAFED A capsules. It is also not known whether
NDVAFED A capsules can cause fetal
arm when administered to a pregnant woman or can affect reproduction capacity, NDVAFED A capsules
may be given to a pregnant woman
only it clearly needer. Pseudoephedrine is contraindicated in nursing
mothers because of the higher than
usual risk for infants from sympathomimetic amines.

ADVERSE REACTIONS: Hyperreachigher land visible and realtive individuals may display.

mimetic amines.

ADVERSE REACTIONS: Hyperreactive individuals may display ephedrine-like reactions such astechycardia, palpitations, headache, dizziness, or nausea. Patients sensitive to antihistamines may experience mild sedation. Sympathosensitive to antihistamines may experience mil seadion. Sympathomimelic drugs have been associated with certain untoward reactions including lear, anxiety, tenseness, restlessness, tremor, weakness, restlessness, termor, weakness, restlessness, termor, weakness, restlessness, and cardiovascular collapse with hypotension. CNS depression, arrhythmias, and cardiovascular collapse with hypotension. Possible side effects of anti-histamines are drowsiness, restlessness, dizziness, weakness, dry mouth, anorexia, nausea, heach, nervousness, blurring of vision, hearburn, dysuria and veraleh, enervousness, blurring of vision, hearburn, dysuria and veraleh, enervousness, blurring of vision, hearburn, dysuria and veraleh graefy to adrenergic agents may be manifested by insomnia, dizziness, weakness, tremor or arrhythmias.

DVERDDSAEE: Acute overdosage with NDVAFED A capsules may produce clinical signs of CNS stimulation and variable cardiovascular effects. Pressor amines should be used with great caution in the presence of pseudoephedrine. Patients

ence of pseudoephedrine. Patients with signs of stimulation should be treated conservatively

treated conservatively.

DDSAGE AND ADMINISTRATION:
DDs capsule every 12 hours. Do not give to children under 12 years of age.

CAUTION: Federal law prohibits dispensing without prescription.

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Merrell Dow

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Prescription relief For the wet symptoms of allergies...

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Capsules

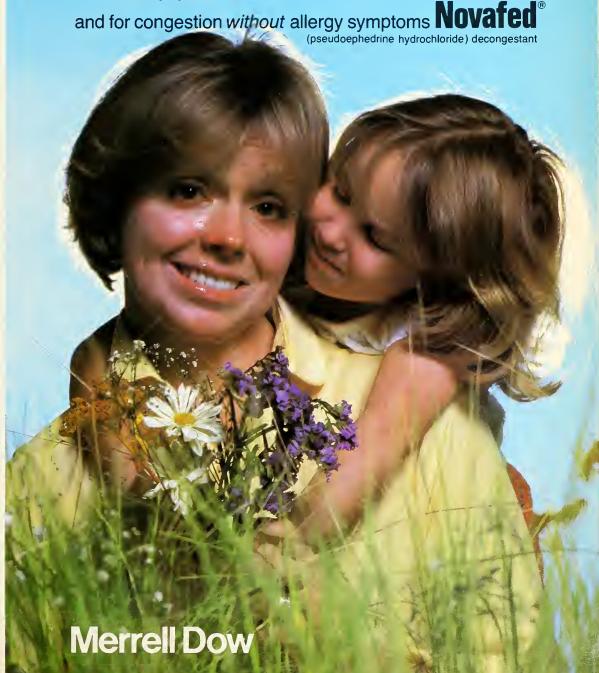
each capsule contains pseudoephedrine
hydrochloride 120 mg, chlorpheniramine
maleate 8 mg

(also available in liquid forms)

Controlled-release decongestant plus antihistamine

Works for 12 full hours

- stops runny nose with decongestant
- relieves allergy symptoms with antihistamine
- dries watery eyes as a result



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Completing Fellowship in ICU/ Ob. Gyn. Trained in all fields including open heart and Epidural Anesthesia.

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NORTHERN MICHIGAN UNIVERSITY located in beautiful Northern Michigan has a position opening for a MEDICAL DIRECTOR — Student Health Center.

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References:

1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. Am Heart J 104.672-681, September 1982

2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. N Engl J Med 302:1269-1273, June 5, 1980

PROCARDIA" (nifedipine) CAPSULES

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nitedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria. 1) classical pattern of angina at rest accompanied by ST segment elevation. 2) angina or coronary artery spasm provoked by ergonovine. or 3) angiographically demonstrated coronary artery spasm in Inhose paties who have had angiography, the presence of significant fixed obstructive disease is not incompatible. with the diagnosis of vasospastic angina, provided that the above criteria are satisfied PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm or when angina is refractory to intrales and or adequate does of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for

the management of chronic stable angina (effort-associated angina) without evidence of viasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents

or who calling tolerate mose agains. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available infor-mation is not sufficient to predict with confidence the effects of concurrent freatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs (See Warnings) CONTRAINDICATIONS: Known hypersensitivity reaction to PROCAROIA

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated occasional patients have had excessive and poorly tolerated. erated hypotension. These responses have usually occurred during initial titration or at the time of ubsequent upward dosage adjustment, and may be more likely in patients on concomitant beta

blockers

Severe hypotension and or increased fluid volume requirements have been reported in patients
receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass
surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be
due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with
PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic
analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose
fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and,
if the patient is condition permits, sufficient time (at least 36 hours) should be allowed for
PROCARDIA to be washed out of the body prior to surgery.
Increased Angina: Occasional patients have developed well documented increased frequency, diation or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased darger and resulting from increased demand
resulting from increased heart rate alone.

increased heart rate alone

resulting from increased neart rate alone. Beta Blocker Withdrawal. Patients recently withdrawal trom beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PRDCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly betore beginning

Congestive Heart Failure: Rarely patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular PRECAUTIONS deneral: hypotension, because PROCANDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCAROIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vaso-diation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with

Peripheral edema. Mild to moderate peripheral edema, typically associated with arterial vaso-diation and not due to let trentricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction. Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional ilterature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination. Origitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve formal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels. It is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of tertility. When given to rats prior to mating, niferine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

man dose Pregnancy Category C Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, inice and rabbits, and abnormalities in monkeys. ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2%, and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCAROIA or concomitant antiangial medication. Additionally, the following have been reported muscle cramps, nervousness, dyspinea, nasal and chest congestion, diarrhea constipation, inflammation, joint stiffness, shakiness, sleep disturbances, burred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever sweating, chilis, and sexual difficulties. Very rarely, introduction of PROCAROIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that soome or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and conges-

than instory of the observer in the patients. In Terhanas possible, indeven, that some of many of these events were drug related. Myocardai infarction occurred in about 4% of patients and conges-tive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturb-ances each occurred in lewer than 0.5% of patients.

ances each occurred in lewer than tip 5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase. CPK. LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nidelpine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCAROIA therapy has been reported twice in the extensive world literature.

HDW SUPPLIED: Each orange, soft gelatin PROCAROIA CAPSULE contains 10 mg of nitedipine PRDCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NOC 0069-2600-41). The capsules should be protected from temperature 59 to 77 F (15°to 25°C) in the manufacturer's original container

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."



"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] as soon as it became available. The change in my condition is remarkable"

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks, taking fewer nitroglycerin tablets,2 doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

*Procardia is indicated for the management of:

1) Confirmed vasospastic angina.

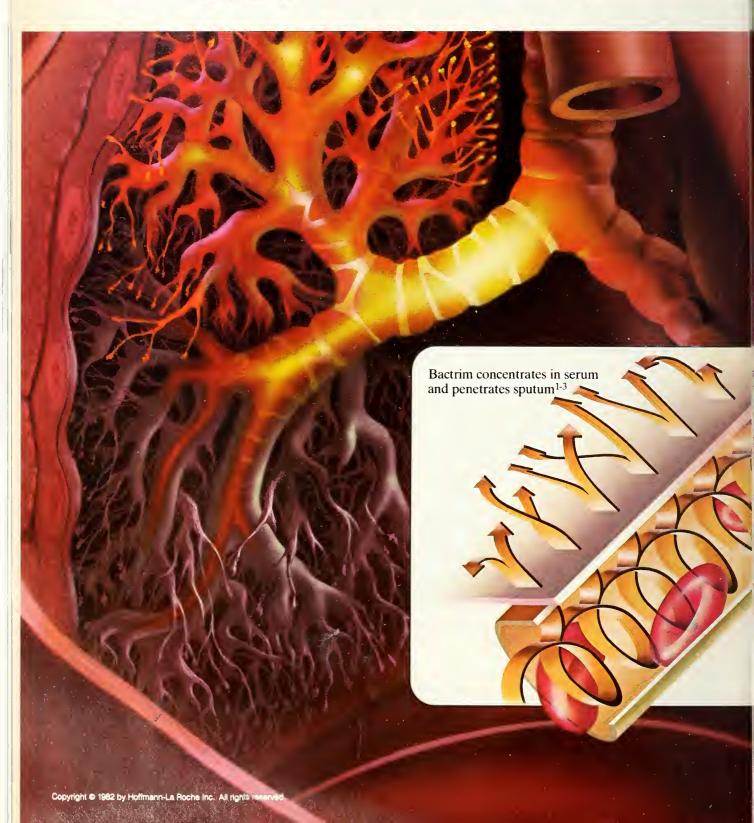
2) Angina where the clinical presentation suggests a possible

vasospastic component.

3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.



Bactrim attacks the (trimethoprim and sulfamethoxazole/Roche) In acute exacerbations





najor pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.^{4,5} One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.⁵

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.⁶

Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters. ⁷⁻⁹ Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

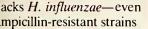
Bactrim also proved more effective than tetracyclines in 10 clinical trials

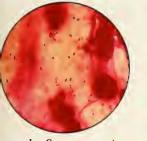
involving nearly 700 patients. ¹⁰ Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: Adv Antimicrob Antineoplastic Chemother 1/2:1105-1106, 1971. 2. Jordan GW et al: Can Med Assoc J 112:91S-95S, Jun 14, 1975. 3. Beck H, Pechere JC: Prog Antimicrob Anticancer Chemother 1:663-667, 1969. 4. Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts; Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. 5. Schreiner A et al: Infection 6(2):54-56, 1978. 6. Data on file, Hoffmann-La Rochc Inc., Nutley, NJ. 7. Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a doubleblind crossover clinical trial, in Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit., pp. 15-16. 8. Chervinsky P: Double-blind clinical comparisons between trimcthoprim-sulfamethoxazole (Bactrim 18) and ampicillin in the treatment of bronchitic exacerbations. Ibid., pp. 17-18. 9. Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. Ibid., pp. 19-20. 10. Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. Ibid., pp. 13-14.





attacks S. pneumoniae



Economical b.i.d.

Bactrim DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

Bactrim

(trimethoprim and sulfamethoxazole/Roche)

Batora praecribing, plaese consult complete product information, a summary of which

Indications and Usaga: For the treatment of urinary tract infections due to susceptible atrains of tha tollowing organisms: Escherichia coil, Kiebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii. It la racommandad that initial apiaodea of uncomplicated urinary tract infactions be treated with a single affective antibacterial agant rather than the combination. Note: The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections. For acuta otitis media in childran due to auaceptibla atraina of Haemophilus influenzae or Streptococcus pneumoniae whan in physician's judgmant it offara an advantaga ovar othar antimicrobiala. To data, there are limited data on the eafety of repeated uaa of Bactrim in childran undar two yaara of age. Bactrim ia not indicated for prophylactic or prolongad administration in otitia madia at any age.

For acuta axacarbations of chronic bronchitis in adults due to auscaptible atrains of Haemophilus influenzae or Streptococcus pneumoniae whan in physician's judgmant it offara an advantaga ovar a singla antimicrobial agant.

For entaritia dua to auscaptible straina of Shigelia flexneri and Shigelia sonnei when antibacterial therapy is indicated.

Also for the treatment of documented Pneumocystis carinii pneumonitia.

Contraindicationa: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL

PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coag-

ulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if poten-

tial benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Allergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. Gastrointestinal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. CNS reactions: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions*: Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goithe production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies. Dosage: Not recommended for Infants lass than two months of age

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis

Children: Recommended dosage for children with urinary tract infections or acute office media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis

For patients with renal impairment. Use recommended dosage regimen when creatinine clearance is above 30 ml/min, If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS.

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. Tablets, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 40. Pediatric Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).



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S Medicine

An Evaluation of Patient Care at South Dakota Human Services Center

Bruce D. Forman, Ph.D* Geraldine P. Lewis, Ph.D.† David W. Bean, M.D.‡

ABSTRACT

A joint effort was initiated in 1977 by the South Dakota Board of Charities and Corrections and the University of South Dakota Board of Regents aimed at improving treatment programs and facilities at the South Dakota Human Services Center. A questionnaire designed to assess

perceptions of change was administered to 30 HSC patients who received care before and after programmatic changes were implemented. Results revealed that changes were perceived by the patients surveyed as positive, suggesting that treatment conditions at the South Dakota Human Services Center are much improved.

Introduction

In 1977 a working relationship was developed between the South Dakota Board of Charities and Corrections and the University of South Dakota Board of Regents with the goal of upgrading the treatment programs and facilities at the South Dakota Human Services Center (HSC) in Yankton, South Dakota. This relationship was initiated by appointing the Chairman, Department of Psychiatry, USD School of Medicine, as the HSC Administrator thereby mobilizing the programmatic and recruitment efforts of the School of Medicine in support of the HSC. This formidable task had several prerequisites, such as recruiting experienced psychiatrists, psychologists and other staff, reorganizing entire treatment programs from a custodial focus to one of

treatment, and the remodeling of patient treatment areas. This paper describes our effort at assessing the impact programmatic changes have had upon patient care at the HSC as viewed by patients in treatment. Patients' perceptions of change were evaluated by a questionnaire developed by USD Department of Psychiatry staff.

Evaluation is an essential element of overall program administration which is concerned with how well policies are translated into organized interventions and the impact on social problems. Program evaluation, according to Suchman, consists of several levels of conception ranging from simply measuring effort (i.e., how many clients seen by clinicians over a set period of time), to complex process/outcome studies (i.e., what procedures have what effects with what groups under what conditions in a given amount of time and at what cost). Measuring effort is perhaps the easiest and least costly method, with associated costs for evaluating other levels of conception and requiring increasingly greater costs depending upon the questions asked.

In mental health service delivery the ideal goal is the immediate access of process and outcome information for every element of services offered

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[†] Assistant Professor, Department of Psychiatry, USD School of Medicine and Clinical Psychologist at SD Human Services Center.

[‡] Professor and Chairman, Department of Psychiatry, USD School of Medicine and Administrator, South Dakota Division of Mental Health.

within a system, including costs per unit, and a direct comparison with elements in a similar system. It may be after the year 2000 that such an evaluative system is even technologically feasible. Many compromises must be made in working toward the ideal evaluation system with cost and available methodology as necessary considerations. A number of useful procedures have already been developed from which inferences can be drawn and managerial decisions based. Among these procedures are studies of service availability, accessability, awareness, length of stay, patient change on objectively scored measures, rates under treatment, cost determination, and acceptability.

Acceptability can be assessed through studies of patient satisfaction. Such studies can also yield an index of treatment outcome since it is a self-assessment of what is happening to the patient. This approach grew in popularity with the consumer movement of the 1970's and is recommended for use in mental health centers and hospitals by no less than the Joint Commission on Accreditation of Hospitals (JCAH). Studying patient satisfaction has the added benefit of allowing patients to view themselves as part of treatment and evaluation processes rather than as objects, with the former view mirroring the current administration's perspective. In addition, this procedure is highly cost effective since it requires a relatively short time to complete.

Design of the Study

Participants: Data were obtained from 30 patients residing in admission, acute and extended treatment areas at HSC. A criterion for inclusion in this study was that a patient must have had another HSC admission prior to 1980 or have been continuously receiving treatment at HSC since at least 1980. One patient's protocol was eliminated due to failure in following directions making results unusable. Units sampled and number of patients included are as follows: Kanner II = 4, Edmunds I = 5, Edmunds II = 5, Edmunds III = 9, Haas II = 6. There were 15 males and 14 females in the sample ranging in age from 19 to 65 years ($\overline{X} = 35.2$ years).

Instrument: A brief instrument which could be completed by patients was constructed to assess changes made in HSC treatment programs. It was administered by HSC ward staff during May and June of 1982. The questionnaire was divided into three sections. The first section listed 15 statements about specific aspects of care. Patients were asked to rate their degree of agreement on the statements about their HSC experience(s) prior to 1980, and for their current admission. A five-point Likert scale was used for ratings. The second section consisted of two open-ended questions and two questions rated

categorically. The third section was answered only by patients who had previously resided on the Kyle, Ordway, or Mellette units. These units were closed prior to the study and represented "old" treatment programs and buildings which had been targeted for improvement. Items in this third section asked for a comparison to be made between perceptions of the current admission experience and prior experience on any of those three units. Data were tabulated and statistically analyzed by USD School of Medicine's computation facility.

Results

Section One: The items in this section were compared via a two-tailed t-test for paired samples on item and total group means. This statistic was selected because scores were derived from one population in which a direction of change was not predicted. The two-tailed test provides a conservative index. For all but one t-test, results were significant beyond the .01 probability level. The mean total scores also differed significantly. All items and total score were higher, indicating perceived improvement, in the current admission. Results of the statistical analysis are shown in Table I.

Section Two: Of the 29 patients included in the sample, 21 had been hospitalized elsewhere for a psychiatric condition at some time in their lives. When asked to compare their care at HSC to that provided by other facilities, only one-third (N = 7)thought the other hospital was better, HSC was rated as better by 6 patients, with 8 patients considering HSC as offering equivalent care. All patients were asked to comment on whether they would recommend HSC to others suffering from mental or emotional problems. Twenty-two patients stated they would recommend HSC; 7 patients would not. Reasons cited for not recommending HSC included going to a private hospital if funds were available, being annoyed by other patients with more serious problems, getting well more quickly in private hospitals, and patients being kept from taking care of themselves resulting in immaturity. This section also included a request for general comments about changes. The vast majority of responses were positive. Specific comments were directed at the living conditions (e.g., open wards, cleaner, more cheerful, co-ed wards, lower census), the staff (e.g., better doctors, more staff), and treatment philosophy (e.g., patients more respected and involved in treatment, isolation used less, classes, and group treatment). There were three negative comments. These pertained to patients having too many activities, not being listened to enough, and receiving little explanation for the presence of prison inmates (trustees) on HSC grounds.

TABLE I
Results of Statistical Analysis for Items in Section One (N = 29)

	Prior Admission		Current Admission			
Items	$\frac{1}{X}$	SD	X	SD	t	p
1. I'm satisfied with the variety			·			
and quality of foods I receive.	3.10	1.24	3.34	1.32	1.32	.199*
2. The nurses and aides are dedi-						
cated to caring for the patients.	3.59	1.24	4.24	.69	3.09	.005
3. I was told enough about my condition.	2.66	1.42	3.72	1.39	4.40	<.001
4. The treatment team has a real interest in me.	3.31	1.28	4.14	.99	3.72	.001
5. I'm treated with respect for my						
judgment and common sense.	3.03	1.35	3.97	.86	3.92	.001
6. Much was done about one or more						
of the problems that brought me here.	3.03	1.24	3.72	1.03	3.36	.002
7. The aides have been helpful.	3.41	1.24	4.21	.62	3.17	.004
8. I'm satisfied with the treat-						
ment I get from my doctors.	3.24	1.24	4.14	.88	3.66	.001
9. There have been enough things						
to do to help pass the time.	3.07	1.16	3.90	.90	3.55	.001
10. My physical health problems have						
been carefully looked into.	3.20	1.47	4.24	1.06	3.55	.001
11. My doctors really understand my problems.	3.28	1.22	4.00	.96	3.27	.003
12. The nurses know their jobs and do them well.	3.24	1.33	4.14	.74	3.39	.002
13. My doctors do their best to keep me from worrying.	3.10	1.24	3.93	.92	3.33	.002
14. I received good care from the psychologists.	3.10	1.18	3.86	1.03	2.91	.007
15. The social worker has been helpful.	3.17	1.26	4.03	.98	3.30	.003
TOTAL	47.62	12.20	59.52	9.15	5.45	<.001

^{*} No significant difference found.

Section Three: This section solicited comments from patients who formerly resided on wards that were decommissioned by the current administration. Responses were received from 13 patients, or 45% of the sample. All respondents agreed that changes at HSC were for the better. Improvements noted were clean and unlocked wards, more time from doctors and other staff, a perception that patients are treated with more respect, encouragement by staff of greater independence and self-responsibility for patients, and an overall sense that conditions are better.

Discussion

Some of the results reported above warrant additional comment. The finding reported for section one that the only item yielding a non-significant result concerned the food service should not be dismissed or treated lightly. In point of the fact, no programmatic effort was deemed necessary to upgrade or change dietary department services since they were considered adequate. Thus, this item serves as a benchmark for other items. That no significant change was observed by patients for something which has not changed contributes to the validity of all questionnaire items. The slightly higher rating patients made on the item pertaining to the

dietary department may reflect a halo effect, but does not invalidate the other items since the magnitude of the significance levels far exceed the required .01 probability level.

It is also worthy of comment to point out that HSC compared very favorably with other psychiatric facilities, especially private ones. This is a high accolade in view of the difference in cost associated with private versus state-sponsored facilities and the generally negative connotation that state hospital treatment has in society.

Summary

A joint effort of the South Dakota Board of Charities and Corrections and the University of South Dakota School of Medicine to improve treatment services at the South Dakota Human Services Center was initiated in 1977. As a result of that joint effort significant improvement in patient care as perceived by the patients actually receiving these benefits has resulted.

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S President's Page



At our upcoming annual meeting in Sioux Falls, our President-elect, Joe Hamm, will take the oath of the president and begin his term. Many of you know Joe, but if you don't, consider making it a point to meet him. He is a true gentleman. Joe has been a valued member of the Executive Committee, Council and Medical Association for many years. His experience and judgement will serve the Medical Association well.

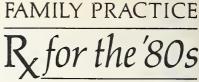
Joe and Dorothy Hamm live in Sturgis, where he was in private practice for years. I know he will represent you ably. Please welcome him and let him know you are his friend.

I want to thank you for the privilege of being your president this year. It was an honor and a privilege. Your cooperation on the many matters that came before us was an inspiration to me.

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Chapter News







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S Medicine

A Practitioner's View of Biomedical Ethics

Jerome W. Freeman, M.D., F.A.C.P.*

ABSTRACT

The scope of biomedical ethics and its implications for clinical practice are considered.

Many physicians are wary of the growing field of biomedical ethics. In general, they tend to view the field either with indifference or hostility. Particularly troublesome is the perception that the medical ethicists' design is to intrude themselves into the physician-patient relationship. The object of this essay is threefold: first, to examine the scope of medical ethics as it is developing in today's society; second, to demonstrate that exploration of medical ethics issues is not destructive of, or intrusive upon, traditional physician-patient interactions; and third, to argue that the arena of medical ethics offers physicians a major challenge to become active participants in the ethical decisions society is making and will inevitably continue to make. Rather than negatively intruding upon the physician's practice, a knowledge and appreciation of the issues of medical ethics places the physician in a position to have a significant impact on societal policies under development.

When considering the range of issues relevant to biomedical ethics, several general categories may be established. The first deals with questions relevant to the dignity and value of human life. In this context, questions dealing with individual autonomy arise. Often these questions focus on issues of informed consent. Other examples include cases in which a patient refuses lifesaving treatment and cases in which treatment decisions for incompetent persons are made. Another series of difficult questions arises when medicine is confronted with pa-

tients in a permanent vegetative state, as typified in the widely publicized Karen Ann Quinlan case. Such cases generate consideration of a possible distinction between human personhood and mere biologic existence. In this context, as in others, issues of euthanasia are often broached. Still other important issues revolve around questions of when life begins, possible fetal rights and abortion.

A second major category of questions may be seen to arise directly from ongoing biologic advances. Examples include the ability to prenatally diagnose genetic defects; the recent development of some types of fetal surgery; in vitro fertilization and the anticipation of future extra-uterine fetal maintenance; the continuing development of artificial organs (most recently the heart); and the emerging prospect of genetic engineering.

A third category of issues may be seen to revolve around global societal concerns. Primary examples are the increasingly urgent questions dealing with the allocation of scarce medical resources and the staggering cost of current and prospective medical technologies. Another example is the question of whether public policy should penalize those who voluntarily take health risks.

Many of these issues and comparable others confront the practicing physician regularly. Moreover, patients and their families almost uniformly expect the physician to advise and participate in such dilemmas. The very frequency with which such issues arise bears testimony to the contention that medicine is, inevitably, an ethical enterprise. To excel in this realm, the physician needs to bring a special expertise to these questions. Certainly this would seem to

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mandate that physicians and prospective physicians receive formal exposure to and training in a reasoned approach to medical ethics issues.

Clearly not all physicians agree with this proposition. Some argue that by the very process of traditional medical education, the physician learns to satisfactorily grapple with biomedical ethics issues. However, common wisdom would suggest that reliance on such a vague "osmosis process" alone is of dubious sufficiency. Since ethical questions and decisions constitute a significant element of medical practice, physicians should bring to these issues an expertise born of formal and careful consideration of ethics and its relation to medical practice. That is, physicians should strive for an ethical expertise that is of the same high caliber as the rest of their medical training.

This point of view is gaining widespread credence. In many medical schools across the country, formal training in medical ethics is provided. Reputable journals and societies devoted exclusively to these issues have been developed. And as a reflection of this movement, numerous articles on medical ethical issues are appearing in general medical journals.

Yet despite this increased awareness of the scope and importance of biomedical ethics issues, many clinicians view the medical ethicist as somehow trying to intrude upon the traditional doctor-patient relationship, and dictate to the physician what clinical decisions are "right." This is a limited and erroneous view. Even in those hospitals which have a medical ethics committee or consulting medical ethicist (and the number of such hospitals is steadily increasing), great pains are taken to avoid dogmatically providing a single "right" answer or prescribed course.² Rather, the procedure at such institutions is to provide a forum in which these difficult ethical questions can be systematically analyzed and considered. Through such consideration, the participants increase their awareness of what ethical principles are involved in given clinical situations and what are the logical ramifications of the clinical decisions they make. Again, such an analysis often does not yield a single correct answer — especially in those cases which pose a conflict between two fundamental principles. But it is through such systematic exposure and analysis that the physician can begin to grasp the full implications of those clinical situations which demand an ethical response. And equally important, such systematic exposure enables the physician to better educate the patient and his family as to various clinical options and ramifications. In this sense, biomedical ethics training is clearly not destructive of the physician-patient interaction. Indeed, it significantly enhances that relationship.

While the foregoing observations have emphasized physician participation in biomedical ethics, many of the issues in this field transcend the isolated clinical setting and are of general concern to all of society. In some measure, society's interest is reflected in the increasing legal interest in many medical ethics issues. Frequently encountered examples include questions of ending care in terminally ill patients; questions of brain death; and issues of decision making for incompetent patients. Another arena of societal concern is reflected in ongoing decisions in this country regarding the proper relationship of government and medicine, especially with respect to cost containment. On this level, wide ranging decisions have been and will continue to be made. Physicians, properly motivated and educated to the issues, are in an excellent position to participate in policy formulation.

In summary, biomedical ethics issues permeate both clinical practice and society as a whole. And in the face of mushrooming biologic and technologic advances, the prospect is that increasingly complex and troublesome ethical conflicts will steadily arise. Physicians have been and will continue to be in a pivotal position with respect to these ethical dilemmas, and will be expected to participate in their resolution. The challenge to physicians is that they develop more than technical medical excellence. Commensurate wisdom and ethical sophistication are also needed. If physicians are not willing to directly confront the complex issues of biomedical ethics, to whom would they defer?

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The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (eg, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used IV: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of band or urist; use extreme care to avoid intra-arrevial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer injectable Valium directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects — particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/dehilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity,

insomnia, rage, sleep disturbances and stimulation have been reported; should these occur discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia. In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: Adults: Anxiety disorders, relief of symptoms of anxiety—Valium (diaze-pam/Roche) <u>tablets</u>, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease <u>capsules</u> (15 to 30 mg) daily. Acute alcohol withdrawal—<u>tablets</u>, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 <u>capsules</u> (30 mg) the first 24 hours, then 1 <u>capsule</u> (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—<u>tablets</u>, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 <u>capsules</u> (15 to 30 mg) once daily. Adjunctively in convulsive disorders—<u>tablets</u>, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 <u>capsules</u> (15 to 30 mg) once daily.

Geriatric or debilitated patients: <u>Tablets</u>—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). <u>Capsules</u>—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg 1.M. or 1.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.) For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary, acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children administer I.V. slowly; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary, in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (IV preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful. In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, IV. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

oral: Valium scored tablets — 2 mg, white; 5 mg, yellow; 10 mg, blue — bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.



SD

This Is Your Medical Association

New officers for the Huron District Medical Society for 1983 are: Wm. G. M. Huet, M.D., president; Ravi Kapur, M.D., vice-president; and Emil Hofer, M.D., secretary. All three physicians practice in Huron

* * * *

The 1983 officers for the Madison-Brookings District Medical Society are: president, Samuel Bandiera, M.D., from Brookings; vice-president, Kim L. Wilde, M.D., from Madison; and secretary-treasurer, Richard Holm, M.D., from Brookings.

* * * *

The seventh District Medical Society recently held their election of officers for 1983. The following were elected: Gail Benson, M.D., president; Rod Parry, M.D., president-elect; Jeff Hagen, M.D., secretary; Jerry Freeman, M.D., treasurer, all of Sioux Falls.

* * * *

The 1983 district officers of the Mitchell District Medical Society are: president, Charles Monson, M.D. of Parkston, vice-president; John Jones, M.D. of Chamberlain; and secretary-treasurer, Timothy Judge, M.D. of Mitchell.

* * * *

Lake Preston has a new physician, **Dr. David J. Halliday**, a native of North Dakota where he has practiced medicine for the past 26 years. Dr. Halliday was born in Kenmore, North Dakota. After graduating from high school, he spent 18 months in the Army before enrolling at the Univ. of North Dakota. He received his medical degree from the Univ. of Nebraska Medical School and interned at St. Luke's Hospital in Fargo, N.D. He did his surgical residency at St. Luke's also. Since that time he has been practicing in North Dakota.

* * * *

Pathologist, Ronald L. Hansing, M.D., has been appointed medical director of St. Joseph Hospital Laboratory. He received his B.A. and M.A. from Southern Illinois University at Carbondale and his medical degree from George Washington Medical School in Washington, D.C. He completed his internship and residency in pathology at the Univ. of California Center for Health Science in Los

Angeles. Dr. Hansing is board certified in anatomical and clinical pathology. He, his wife and two children moved to Mitchell from Visalia. CA.

* * * *

Michael Sperl, M.D. has opened his practice of neurology and neurosurgery in Watertown. Dr. Sperl came here from St. Paul, Minnesota where he has practiced for 23 years. He graduated from the Univ. of Minnesota Medical School and did his internship at the King County Hospital, Seattle, Washington. He received his neurosurgery training at the Mayo Foundation in Rochester, Minnesota. He is a fellow of the American College of Surgeons and a diplomat of the American Board of Neurological Surgery.

* * * :

David R. Rossing, M.D., a specialist in pulmonary disease in Sioux Falls, has been elected to fellowship in the American College of Physicians.

* * *

Christopher Moller, M.D. of Mitchell was elected to a three year term on the St. Joseph Hospital Board of Trustees at their annual meeting. He replaces Donald Weatherill, M.D. who served two three-year terms on that Board.

* * * *

The 1983 officers for the Watertown District Medical Society are: Richard McClaflin, M.D., Watertown, is president; James Horning, M.D., Watertown, is vice president; and Gerald Tracy, M.D., Watertown, is secretary/treasurer.

YOUR CONTRIBUTION
IS NEEDED TO THE
SOUTH DAKOTA
MEDICAL SCHOOL
ENDOWMENT FUND

Dr. Larry Ebbert, Rapid City spoke on what cancer is, its causes, types, locations and methods of treatment at a meeting held in Rapid City by the Rapid City Regional Hospital.

* * * *

Two Sioux Falls physicians have been elected to fellowship in the American College of Cardiology. They are: Paul L. Carpenter, M.D. and Mary Slattery, M.D.

* * * *

Sioux Falls pathologist, **Loyd Wagner**, **M.D.**, was re-elected to a two year term as vice speaker to the House of Delegates of the College of American Pathologists.

* * * *

Arthur Lampert, M.D., Madison, was elected medical advisor of the South Dakota Chapter National Multiple Sclerosis Society at their annual meeting.

* * * *

John G. Langdon, M.D. of Sioux Falls was elected to a two year term on the Board of Directors of the recently formed National Wellness Organization, headquartered in Stevens Point, Wisc.

* * * *

J. M. McMillan, M.D. of Sioux Falls was elected president of the S. D. Division of the American Cancer Society at its annual meeting held in Sioux Falls.

* * * *

Dr. A. J. Tieszen, Pierre, was re-elected president of the Pierre Rural Area Health Education Center (AHEC) Board of Directors at their meeting.

* *

Rapid City physician, H. Benjamin Munson, M.D., was presented with the American Civil Liberties Union's first humanitarian award at the ACLU state convention in Sioux Falls.

* * * *

A retired Sioux Falls physician, R. B. Leander, M.D., was elected president of the Presentation Health System Foundation.

John Jones, M.D. of Chamberlain and Michael Justice, M.D. of Dell Rapids have been named diplomates of the American Board of Family Practice.

* * * *

Jerome Eckrich, Jr., M.D., Aberdeen, has been named Fellow of the International College of Surgeon.

John Davis, M.D., Pierre, has been elected to serve on the Maryhouse Governing Board for two years.

* * * *

Samuel Heth, M.D., Mitchell, has been certified by the American Board of Obstetrics and Gynecology.

* * * *

Timothy Judge, M.D., Mitchell, has successfully completed the board examinations and is now certified as a diplomate of the American Board of Internal Medicine.

* * * *

Dr. Anthony Salem, Sioux Falls, was elected president of the Minnehaha Unit, Dakota Affiliate of the American Heart Association and **Dr. Charles O'Brien,** Sioux Falls, was elected vice president.

* * * *

Four South Dakota physicians have attained Fellowship status in the American College of Chest Physicians. They are: **Drs. Lewis Offstein, Leycester Owens, James Reynolds** and **David Rossing** all of Sioux Falls

RISK MANAGEMENT SEMINAR

June 3 & 4, 1983

Ramada Inn Sioux Falls, SD





Prompt, effective relief from the dual burden of pain and anxiety

Equagesic-M

(meprobamate with aspirin) © Wyeth Effective analgesic/anxiolytic alliance

Proven superior to aspirin alone in controlled clinical trials

(BRIEF SUMMARY)

DESCRIPTION: Each tablet contains 200 mg meprobamate and 325 mg asplifin. INDICATIONS: Adjunct in short-term treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease. Clinical trials demonstrated in these situations relief of pain is somewhat greater than with aspiral and extension and the contained of th

drug for Individual patients. CONTRAINDICATIONS: ASPIRIN: Al-

should periodicially reassess usefulness of drug for individual patients.

CONTRAINDICATIONS: ASPIRIN. Allergic or idlasyncratic recordions to aspirate the recording to relative the recording to the recording

within next 12-to-48-hour period When excessive dosage has continued for weeks at months, reduce dosage gradually over 10-2 weeks rather than stop abruptly. Alternatively, a short-acting parbiturate may be substituted, then gradually withdrawn. FOTENTIALTY HAZARDOUS TASKS: Warn patients meprobamate may impair mendor physical abilities required far potentially hazardous tasks, e.g., affixing or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caulion with patients taking more than one of these agents simultaneously

ercise caulion with patients toking more than one of these agents simultaneously USASE IN PREGNANCY AND LACTA. IION: An increased risk at cangenital malformatians assaciated with minor tranquillizers (meprobamate, chicral-azepaxide, and diazepam) during first itimester of pregnancy, has been suggested in several studies. Because used in these areas is used to the second of the

about destrability of discantinuing the drug.

Meprabamate passes the placental barrier. It is present both in umbilical-card blacd at an ear moternal plasma levels and in breast milk at lactating mathers at cancentralians who to laur times that of maternal plasma. When use at meprabamale is cantemplated in breast leeding plasma. When use at meprabamale is cantemplated in breast entitle of cancentrations in breast milk as compared to maternal plasma levels. USAGE IN CHILDREN. Keep preparations with aspirin out of reach of children. Equagesic*. Mis not recommended for patients 12 years of age and under. PRECAUTIONS: ASPIRIN: Sallcylates an-

fagonize uricosuric activity of probene-cid and sulfinpyrazone. Salicylales are reported to enhance hypoglycemic ef-fect of sulfonylurea antidabetics. MEPROBAMATE: Use lowest effective dose, parficularly in effety and/or debil-itated, to preclude over-sedation. Me-probamate is metabolized in the liver and excreted by the kidney, to avoid ex-cess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occa-sionally may precipitate seizures in epi-leptic patients. It should be prescribed caullously and in small quantifies to pa-tients with suicidal tendencies.

cauliously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS: "ASPIRIN: May cause epigastric discomfort, nausea, and vomiting, Hypersensitivity reactions, including urlicaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Potients receiving large doses of salicylates may develop tinnitus.

laxis may rafely occur. Potients receiving large doses of salicylates may develop tinntus. MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impalmient of visual accammodation, euphoria, overstilmutation, paradoxical excitement, tast EEG activity. GI Nausea, vomiting, diarrhea, CARDIOVASCULAR: Palpitation, tachy-carbonates, and the compassion of th

bullous dermatitis have occurred HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCATIC"): Agranulocytosis, aplastic anemia have been reported, although na causal relationship has been established, and thrambacytapenic

purpura. OTHER Exacerbation of porphyric

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Nat recommended for patients 12 years of age and under

OVERDOSAGE: Treatment is essentially ÖVÉRDOSAGE. Treatment is essentially symptomatic and supportive Any drug remaining in the stomach should be removed Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of sailcylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorthagic manifestations due to hypoprothrombinemia which, if il occurs, usually requires whole-blood due to hypoprothombinemia which, if It occus, usually requires whole-blood transfusions. Suicidal artempts with meprobamate have esuited in drawsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been statal. The fallowing data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as liftle as 12 gram meprobamate and survival with as much as 40 gram.

BIOOD LEVELS:

BLOOD LEVELS:

0.5-2 0 mg percent represents usual blood-level range after the apeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to

lindings of mild-to-moderate symptoms of overdosage, such as stupor or light

Indings of mild-to-moderate symptoms of overdosage, such as stupor or light coma 10-20 per cent usually corresponds to deeper coma, requiring more intensive feediment. Some farbilities accurrent, more fatalities from survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or fissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory are some compromised, and the survivals and pressor agents should be administrative and pressor agents should be administrative and pressor agents should be administrated cautiously as indicated. Diuresis, somolic (mannifol) diuresis, perionead idalysis, and hemodallysis have been used successfully in removing both aspiring and hemodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring and memodallysis have been used successfully in removing both aspiring have a memoral and advantage and deathy. Administration of the urine increases excretion of salicylates. Careful monitoring of uninary output is necessary, and caution should be affected to the properties of the properties of the properties.

c 1983, Wyeth Laborataries



S Medicine

Accidental Use of Superglue in the Eye

J. Kemper Campbell, M.D.*

The hazards of mistaking similarly packaged ophthalmic drugs have been previously reported.^{1, 2} Similar confusion with a nonophthalmic compound is now described.

Case Report

A 35-year-old man was working outdoors April 24, 1982, when his eyes began to itch. He found a bottle of what he believed were his wife's eyedrops on the windowsill. Immediately upon placing one drop into his right eye, he experienced the onset of severe pain in the eye and an inability to open his eyelids. Inspection of the bottle revealed that he had mistakenly instilled Superglue into the eye.

He was taken to the local emergency room where the examining physician was unable to visualize the cornea because of the firm lid adhesion present.

He was seen at the St. Elizabeth Community Health Center emergency room approximately three hours after his initial injury. His right lids and lashes were firmly bonded together by the adhesive. Under magnification, surgical scissors were used to carefully separate his lids and remove all lashes.

As soon as the lids separated, a gush of tears and debris which had been trapped behind the lids exited. A large central corneal abrasion was then noted. A clump of dehisced corneal epithelium and Superglue was present in the inferior conjunctival fornix.

* 630 No. Cotner, Suite 105, Lincoln, NE 68505.

Pressure patching, topical antibiotics, and cycloplegic drops healed the abrasion, though the patient continued to be quite uncomfortable for the next 72 hours

When last seen May 6, 1982, the patient again had 20/20 corrected vision acuity and his cornea was completely clear.

Discussion

Superglue is a commonly used adhesive compound which is found in many households. Various nonprescription eyedrops are packaged in similarly sized and shaped bottles which can potentially be confused with Superglue.

As physicians we should be aware of potential environmental hazards to our patients, and if we do occasionally recommend eyedrops which can be purchased over the counter, we should emphasize that the proper place for these drops is the medicine cabinet.

It is also our responsibility to alert the manufacturers of this possible confusion with their product so that a simple change in packaging (i.e. using a red label) may be considered to prevent further accidents of this type.

REFERENCES

- 1. Partamian L and Kass M: Similar packaging of ophthalmic drugs. Am J Ophthalmol 92:586, 1981.
- Katz N and Foer E: Ophthalmic drugs with similar packaging. Am J Ophthal 93:253, 1981.



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> > **PHONE 336-3818**

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South Dakota Society Of **Pathologists**

Officers for 1982-83

Jerry L. Simmons, M.D., President Thomas E. Henry, M.D., Vice President Beth L. Johnson, M.D., Secretary-Treasurer



S Auxiliary News

1982-1983 AUXILIARY INVOLVEMENT

South Dakota Auxiliary involvement was apparent when Marie Hoyland (James), president-elect delegate, and I attended the 60th annual AMA Auxiliary convention held at the Drake Hotel in Chicago, June 13-16, 1982. South Dakota Auxilians received two AMA-ERF awards. The first award was for the state auxiliary in the North Central region with the largest contribution per capita, \$50.96. THIS WAS SECOND FOR THE ENTIRE NATION. Also, for the third consecutive year, District 1. Aberdeen led the North Central region and received an award for the largest per capita contribution of \$122.59. South Dakota physicians and spouses contributed a total of \$24,463.47 to AMA-ERF last year, through fund raising events such as auctions, home tours, sale of Christmas cards, cookbooks, and many numerous items donated from each district to the boutique at the annual state convention, plus personal donations. Kay Reaney (Duane), State AMA-ERF Chairman, with 100% district participation, anticipates another successful year for AMA-ERF.

South Dakota also received a membership award for a 10 percent increase within the North Central region. Membership, a vital tool in any organization was promoted this year by State Membership Chairman, Jeanne Taylor (Wm.), as was the continuation of the Sponsor a Spouse Program allowing the medical student spouses membership in the AMA Auxiliary, thus building for the future.

More great news. Not since 1980 has South Dakota had a representative on a National AMA Auxiliary committee. This year Ila Lushbough (Bruce), so capably serves on the National AMA-ERF Committee and is in charge of Christmas cards and Holiday Sharing cards.

The South Dakota State Medical Auxiliary conducted a Fall "mini" Workshop, during the second Board of Directors meeting, held in Sioux Falls on September 29. Samples of auxiliary materials that are available from the AMA Auxiliary headquarters were on display for AMA-ERF, Health Projects, and Membership. District Presidents and State Chairmen reported on auxiliary projects throughout the state. Speakers included North Central Region Representative Mary Kay McPhee (Wm.), AMA-ERF Committee member, Ila Lushbough (Bruce), and Health Education, Dr. David Paulsrud, Sioux City,

Iowa.

October found six South Dakota Auxilians traveling to the Fall AMA Auxiliary Confluence in Chicago, October 9-11. We returned filled with new ideas and enthusiasm to share with others throughout the state

There are two special projects that deserve recognition. They are in Rapid City and Sioux Falls. District Nine's "Ellie and Her Girls" for their generous volunteerism for AMA-ERF. They have appeared in Rapid City for several functions, Brookings, the North Central Conference in Minneapolis, MN, Watertown, Aberdeen, Yankton, and destinations yet unknown. Also a big thanks to District Seven and the energetic efforts of Myrna Anderson (Courtney), JoAnn Morris (Alan), and the many volunteers who helped make the Volksmarch a success.

Auxilians are becoming involved in legislation. Forty-four auxilians and legislators gathered for dinner during our annual Auxiliary Legislative Day in Pierre. The event, planned by Ellen Kunz (James), State Legislation Chairman, provided auxilians an opportunity to familiarize themselves with the process of state government, get acquainted with our legislators and legislative issues.

State Health Projects Chairman, Mary Bandiera (Sam) has encouraged the Shape Up for Life campaign and is conducting a state wide health poster contest, wherein each district can participate and winning posters will be displayed for final judging at the state convention.

Auxiliary members continue to contribute volunteer hours to community projects, AMA-ERF, public relations, and legislative awareness on health and medical issues.

This being my final communication as my year as auxiliary president draws to a close, I would like to take this opportunity to thank the South Dakota State Medical Association for kindly allowing me this space in the **Journal** to convey the auxiliary message.

Mrs. Richard I. Porter (Marlys)
South Dakota State Medical Auxiliary President

Marlip Tox

S Future Meetings

June

- Interdisciplinary Approach to the Treatment of the Critically Ill Patient, Saint Paul Hotel, St. Paul, MN, June 9-11. Fee: \$175. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101.
- The Sixth Annual Black Hills Seminar on Advances in Clinical Pediatrics, Sylvan Lake Resort, Custer, SD, June 22-24. Contact: Lawrence R. Wellman, M.D., Dept. of Ped., USD School of Med., P.O. Box 5039, Sioux Falls, SD 57117-5039. Phone: (605) 333-7178.
- National Behavioral Pediatrics Conference, Earle Brown CME Ctr., St. Paul, MN, June 23-25. Fee: \$300. 22 hrs. AAFP, AAP, APA & AMA Category I credits. Contact: Lori Wheatcroft, Regis., CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.
- Human Aging VI, 125 Willey Hall, U. of Minn., Minneapolis, MN, June 29-30. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

July

- CME Alaskan Cruise/Conference on Legal-Medical Issues, Pacific waters on board TSS FAIRSEA, July 2-16. 24 hrs. AMA Category I credits. Contact: Linda Wynn, International Conf., 189 Lodge Ave., Ste. C, Huntington Station, NY 11746. Phone: (516) 549-0869.
- CME Caribbean Cruise/Conference on Legal-Medical Issues, Caribbean waters on board TSS FAIRWIND, July 27-Aug. 6. 24 hrs. AMA Category I credits. Contact: Linda Wynn, International Conf., 189 Lodge Ave., Ste. C, Huntington Station, NY 11746. Phone: (516) 549-0869.
- The Total Hip: Current Status, Hyatt Regency Hotel, Minneapolis, MN, July 28-30. Fee: \$350. 17 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Medical Problems of Musicians Conference, Aspen Musical Festival, Aspen, CO, July 27-Aug. 1. 20 hrs. AMA Category I credits. Contact: Aspen Music Festival, 1860 Broadway, #401, New York, NY 10023. Phone: (212) 581-2196.

August

- 1983 Black Hills Summer Seminar, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 11-13. Fee: \$100. 15 hrs. AAFP & AMA Category I credits. Contact: L. H. Amundson, M.D., 3001 S. Holly, Sioux Falls, SD 57105. Phone: (605) 335-5008.
- Annual Sixth District American College of Obstetricians and Gynecologists Meeting, Rushmore Plaza Civic Ctr., Rapid

City, SD, Aug. 18-20. Contact: The Women's Clinic, 2805 Fifth St., Ste. #110, Rapid City, SD 57701. Phone: (605) 343-6550.

CME Mediterranean Cruise/Conference on Legal-Medical Issues, Mediterranean waters on board MTS DANAE, Aug. 20-Sept. 3. 24 hrs. AMA Category I credits. Contact: Linda Wynn, International Conf., 189 Lodge Ave., Ste. C, Huntington Station, NY 11746. Phone: (516) 549-0869.

September

Atlantic-Mediterranean Meeting on Gastroenterology, Mediterranean Cruise, Sept. 17-Oct. 7. Fee: \$1,640 includes passage, meals, and other extras. Contact: Mario Blanco Peres, Rua Goncalo, Cristovao, 116-3°, 4000 Porto, Portugal. Phone: 24294, 20933 or 31002. Telex: 26850 FRZDN-P.

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not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

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Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

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ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

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INJECTABLE To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used IV: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small reins, i.e., dorsum of band or urist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valum directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence, can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhihitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity,

insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia. In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported. Dosage: Individualize for maximum beneficial effect.

ORAL Adults: Anxiety disorders, relief of symptoms of anxiety—Valium (diaze-pam/Roche) <u>tablets</u>, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease <u>capsules</u> (15 to 30 mg) daily. Acute alcohol withdrawal—<u>tablets</u>, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 <u>capsules</u> (30 mg) the first 24 hours, then 1 <u>capsule</u> (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—<u>tablets</u>, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 <u>capsules</u> (15 to 30 mg) once daily. Adjunctively in convulsive disorders—<u>tablets</u>, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 <u>capsules</u> (15 to 30 mg) once daily.

Geriatric or debilitated patients: <u>Tablets</u>—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). <u>Capsules</u>—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to $2\frac{1}{2}$ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE. Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.) For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg 1.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults. 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children administer I.V. slowly; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful. In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

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INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% henzyl alcohol as preservative.



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S Medicine

The Role of Tracheostomy in Acute Laryngeal Obstruction in Children

Thomas G. Bunker, M.D.* Winston B. Odland, M.D.†

ABSTRACT

The management of acute laryngeal obstruction in children has been recognized as a challenging problem for over fifty years. The development of methods of treatment over this time period has lowered the mortality rate from about

In 1978, Francis W. Davison¹ presented a scholarly paper on "Acute Laryngeal Obstruction in Children, a Fifty Year Review." This presentation based on a thorough review of the literature and Doctor Davison's extensive personal experience with this subject clearly pointed out the methods that have in the past fifty years reduced the mortality rate from about seventy per cent to nearly zero. Before reviewing the development of these methods it is necessary to have a classification and description of the major diseases producing this disorder. We prefer the classification system proposed by Davison² in 1950 which is as follows:

- 1. Acute laryngo-tracheal bronchitis (subglottic laryngitis, non-diphtheritic croup)
- 2. Acute supraglottic laryngitis (acute epiglottitis)
- 3. Laryngeal diphtheria (diphtheritic croup)
- 4. Supraglottic allergic edema (angioedema of the larynx)

seventy per cent to almost zero. The controversy between the choice of tracheostomy or nasal tracheal intubation must be considered relative to the facilities and personnel available in the various hospitals where these problems are treated.

- 5. Subglottic allergic edema (spasmodic laryngitis, nocturnal croup)
- 6. Foreign body in the larynx

Since our subject in this presentation is the role of tracheostomy in acute laryngeal obstruction in children we will concern ourselves primarily with the first two diseases in this classification system and mention the last four here only briefly.

Laryngeal diphtheria, while always mentioned, is rarely seen anymore. Management of the airway in this disorder is essentially the same as in laryngo-tracheal bronchitis with the exception that repeated bronchoscopic removal of crusts is often necessary. The medical treatment of diphtheria can be found in any standard textbook.

Supraglottic allergic edema may be due to hypersensitivity to drugs, food, inhalants or horse serum. A pale watery edema of the epiglottitis and aryepiglottic folds develops rapidly causing airway obstruction. This disease responds quickly to intravenous steroids or subcutaneous epinephrine.

Subglottic allergic edema or nocturnal spasmodic croup is felt by many authors to represent an allergic disorder. Others suggest a viral etiology. It may be that both etiologies can be present in this disorder either alone or in combination. Whatever the etiolo-

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gy this disorder is the common night croup which is seen frequently by all physicians who treat a significant pediatric population. This disease responds rapidly to high humidity, steroids, and/or racemic epinephrine.

The two major causes of serious acute laryngeal obstruction in childhood are acute epiglottis and acute laryngotracheal bronchitis.

Tracheostomy or intubation should be rarely if ever necessary. It is important to distinguish this disorder from acute laryngo-tracheal bronchitis both from the standpoint of immediate treatment and for the purposes of significant statistics. For example, spasmodic croup responds quite readily to racemic epinephrine, whereas in acute laryngo-tracheal bronchitis, steroids, antibiotics and preparation for possible airway intervention are indicated. Studies, where concise diagnosis has not been made, have produced statistics that would tend to encourage clinicians to rely on racemic epinephrine in cases where a more aggressive approach is indicated.

Tracheal and laryngeal foreign bodies should be considered in cases of acute laryngeal obstruction. A high index of suspicion and a proper endoscopic examination will lead to the appropriate resolution of this problem.

The two major causes of serious acute laryngeal obstruction in childhood are acute epiglottitis and acute laryngo-tracheal bronchitis. Acute laryngotracheal bronchitis occurs primarily in children under three years of age. The edema and inflammation involves the vocal cords and subglottic area and may extend downward throughout the tracheal bronchial tree. It does not involve the structures above the vocal cords. The symptoms, beginning with a croupy cough and a hoarse voice, usually have a gradual progression leading to inspiratory and expiratory stridor and, in some cases, upper airway obstruction. There is usually no dysphagia or drooling. The edema and inflammation in the subglottic area is the direct cause of airway stenosis and obstruction. This diagnostic term was described by Baum³ in 1928 who discussed his series of twenty eight cases, ten of which died. At that time the O'Dwyer intubation tube was used. In the late 1930's the Jacksons, 4 Hollinger⁵ and others advocated humidified air, parenteral fluids, and early tracheostomy instead of intubation. The mortality rate dropped to around twenty five per cent. In 1948 Davison⁶ reported fifty-two consecutive cases fifteen of which required tracheostomy with no deaths. These patients received either sulfadiazine or penicillin along with humidified oxygen, careful personal attention and tracheostomy as indicated and, at times, blood transfusions. In 1967, Davison⁷ reported one hundred fourteen cases, eighty per cent of which received IM hydrocortisone injections. There were no tracheostomies and no deaths in this series which gives very strong support to the effectiveness of steroids in treating this disorder. In 1974 Schuller and Brick⁸ presented a study including 815 cases of laryngo-tracheal bronchitis and concluded that nasotracheal intubation was safer than pediatric tracheostomy in treating the cases which required mechanical airway maintenance. There were no fatalities and delayed irreversible complications occurred in only 1.6% of the patients intubated.

Acute supraglottitis, more commonly referred to as acute epiglottitis, occurs most frequently in preschool age children. The course of this disease is shorter and more threatening than that of laryngotracheal bronchitis. The duration between early upper respiratory infection symptoms and laryngeal obstruction averages less than twelve hours and may be as little as two hours. The inflammatory edema involves the supraglottic structures including the epiglottis, the aryepiglottic folds and arytenoid area.

The reports comparing tracheostomy and nasotracheal intubation contain too many variables to be statistically valid. Either method seems to be highly effective in managing this disorder under optimal conditions.

The vocal cords and subglottic area are not significantly involved. The voice may have a muffled quality but marked hoarseness and croupy cough are generally not present. Dysphagia, drooling and inspiratory stridor are the prominent symptoms. Acute obstruction may occur suddenly especially during the examination or attempts at intubation. In 1967, Baxter⁹ reported one hundred and three cases of acute epiglottitis, one hundred of which required tracheostomy. There was one death in this series. Fearon¹⁰ in 1977 reported on one hundred cases and noted a decrease in tracheostomy rate from eightyfive per cent in 1970 to fifty per cent in 1975. This was attributed to earlier diagnosis with vigorous medical therapy. There were no deaths in this series. At the same time Fearon and Bell¹¹ were asked by the Ontario Medical Association to investigate a number of deaths occurring in the Province in patients with acute epiglottitis. They concluded that these deaths were due to delayed diagnosis and unsuccessful attempts at endotracheal intubation. Schuller and Birck's 12 series included fifty five patients with epiglottitis, sixty per cent of whom required intubation. General anesthesia usually with a

relaxant was used for intubation. Intubation was possible in every case but five per cent of these later required tracheostomy. There was no mortality in this series. A review of the literature has revealed several reports of cases where intubation was unsuccessful; in some instances resulting in respiratory

Given the conditions existing in the average community hospital such as those in many of the larger towns in South Dakota, it is our opinion that nasotracheal intubation presents increased difficulties and potential complications when compared to tracheostomy in dealing with acute laryngeal obstruction in children.

arrest. Similarly, several published series have reported a higher rate of complications and mortality with tracheostomy than the ones reported in this paper. The reports comparing tracheostomy and nasotracheal intubation contain too many variables to be statistically valid. Either method seems to be highly effective in managing this disorder under optimal conditions. Optimal conditions for nasotracheal intubation would include the following:

Initial evaluation and treatment by a team including the pediatrician, an otolaryngologist and an anesthesiologist skilled at nasotracheal intubation in children. The otolaryngologist must be present and prepared to immediately insert a pediatric bronchoscope and perform a tracheostomy if attempted intubation is unsuccessful. A Pediatric Intensive Care Unit with 24 hour in-service physician coverage is an important part of this optimal situation. Most important is the necessity for constant observation by a nursing staff experienced in the care of small children with nasotracheal tubes. Momentary lapses in this observation frequently result in accidental extubation.

While these optimal conditions obviously exist in the large medical centers where nasotracheal intubation appears to be a safe and effective means of treating acute laryngeal obstruction in children, it is not realistic to expect to find them all in the average community hospital. Given the conditions existing in the average community hospital such as those in many of the larger towns in South Dakota, it is our opinion that nasotracheal intubation presents increased difficulties and potential complications when compared to tracheostomy in dealing with acute laryngeal obstruction in children. Our reasons for this opinion are as follows:

First, there is the basic patho-physiologic problem of maintaining an endotracheal tube in an in-

flamed larynx. Hollinger¹³ in a study in 1965 reported these findings in infants intubated for a variety of non-inflammatory conditions. Mucosal congestion occurred in 2-4 hours, ulceration in 6-8 hours, and cartilage involvement in 30-48 hours. Even if the use of steroids might minimize the added burden of inflammatory edema in these cases, we still regard this as a matter of some concern. Of even more concern are the problems of mechanics of intubation, maintenance of the endotracheal tube and eventual extubation. There are cases where either nasal or oral intubation with an endotracheal tube are extremely difficult and at times impossible. This has been reported in the literature and confirmed in our own experience. In acute supraglottic laryngitis the inflammatory edema of the supraglottic structures may prevent visualization of the vocal cords with a Flagg laryngoscope especially in the child who is awake and breathing vigorously. In these cases introducing the pediatric bronchoscope thorugh a Jackson type slide laryngoscope will allow visualization of the cords and intubation with a minimum of trauma. In cases of severe subglottic edema it may be quite difficult to insert even a small rigid pediatric bronchoscope and would be impossible

Restlessness has been interpreted as a probable sign of hypoxia indicating a need to check the airway rather than to sedate the patient.

to pass a flexible endotracheal tube. If nasal tracheal intubation is successfully carried out one is then faced with the problems of maintaining it. To prevent accidental dislodgment of the endotracheal tube, constant close nursing care observation is necessary along with the use of restraints and fairly heavy sedation. One study¹⁴ recommended alternate administration of morphine and diazapam intravenously every four hours. With tracheostomy we seldom find the restraints necessary. We have traditionally avoided the use of sedation. Restlessness has been interpreted as a probable sign of hypoxia indicating a need to check the airway rather than to sedate the patient. The possibility of accidental dislodgment of the endotracheal tube in a sedated patient is even more frightening. We are also concerned with the effect of depressing the cough reflexes with sedation in these patients. This is especially important in view of the relatively high incidence of atelectasis and/ or pneumonia in these cases. Obstruction of the endotracheal tube may be more difficult to deal with than in the case of a tracheostomy where the inner cannula can be easily removed and cleaned.

Extubation is usually a more complicated procedure with an endotracheal tube than it is with a tracheostomy. Although, as in two cases we were following, the patients may at times successfully extubate themselves. It is usually recommended that direct laryngoscopy be done immediately before and after extubation to estimate the adequacy of the airway. Tracheostomy on the other hand may be corked for 24 hours to demonstrate the adequacy of the airway prior to removal. We have not seen any problems decannulating these children over the years.

Of paramount importance is early diagnosis and airway assessment.

Based on these factors we would recommend the following approach to the treatment of acute laryngeal obstruction in children. Of paramount importance is early diagnosis and airway assessment. In cases of severe respiratory distress, especially where the symptoms would suggest an acute epiglottitis, vigorous examination of the patient's throat should be avoided. We would also recommend deferring X-ray and laboratory study in such cases and proceeding immediately to the operating room to establish a diagnosis by direct examination and proceed with tracheostomy. In milder cases, we would recommend a short course of medical therapy under direct observation. By direct observation, we mean with the laryngologist in constant attendance, and all preparations for bronchoscopic intubation and tracheostomy made. In a case of suspected epiglottitis this period of observation would generally be between 30 minutes and one hour. The treatment we would recommend at that time would be high humidified oxygen administration along with a short course of steroids in full dosage. Intravenous fluids should be given to correct any dehydration and as a route of administration of medications. Antibiotic treatment should be started particularly directed at the hemophilus influenzae. Racemic epinephrine may be of some help in cases of acute laryngotracheal bronchitis but should not be relied upon in acute epiglottitis. If there is no improvement in the airway condition over this period of time we would proceed to the operating room. Preliminary oral endotracheal intubation may be attempted at this time if preferred but all preparations should be made for immediate insertion of a pediatric bronchoscope if the intubation is unsuccessful. Once the airway is provided with an endotracheal tube or a bronchoscope we would proceed with the tracheostomy. We prefer the classic Jackson type tracheostomy with the short vertical incision. We have found the potential problems of a transverse incision out-weigh the minimal cosmetic problems of a vertical incision. We generally use a metal type Jackson tracheostomy

tube with an inner cannula. Special care must be taken during the surgical procedure to avoid injuring the dome of the pleura which could result in a pneumothorax. Immediately following the tracheostomy a chest X-ray should be done along with careful auscultation of both sides of the chest to rule out a pneumothorax and check the placement of the tracheostomy tube. Highly humidified oxygen should be administered either by a humidity tent or a nebulizer shield. It is well to spend some time orientating the Intensive Care nurses regarding pediatric tracheostomy care at this time. Medical treatment is then continued as outlined before. After forty-eight hours we generally try to change the tracheostomy tube and replace it with a size smaller. If this is well tolerated, the tube is corked for twenty-four hours and then removed.

In summary, over the years, we have seen that the principles and methods of treatment for acute laryngeal obstruction in children have progressively developed to the point where mortality and morbidity should be minimal. Nasal tracheal intubation appears to be a reasonable alternative to tracheostomy as a means of airway maintenance when performed under the optimal conditions present in many large medical centers. We feel it introduces some additional, unnecessary risks when attempted in the average community hospital. Under these conditions we still believe that tracheostomy is the keystone to airway management in these acutely ill children.

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ercise coulion with patients taking more than one of these agents simultoneously. USAGE IN PREGNANCY AND LACIA-IION. An Increased itsk of cangenital mallarmations associated with minor tranquillizers (meprobamate, chlordiazepoxide, and diazepam) during first itemsets of pregnancy, has been suggested in several studies. Because use of the several studies, because use of the several studies, because use of the several studies. Because use of the several studies, because used has a several studies, and the several studies and the several studies. Because used has a several studies, and the several studies are several studies, and the several studies are several studies. Because used in the several studies, and the several studies are several studies. Because used in the several studies are several studies, and the several several

about desirability of discontinuing the drug Meprobamate passes the placentat barrier. It is present both in umbilical-cord blood at an near maternal plasma levets and in breast milk of lactalting mothers at concentrations who to four times that of meternal plasma. When use of meprobamate is contemplated in breast fielding mothers are desired in the contemplated in breast fielding before the cancentrations in breast milk as compared to maternal plasma levets. USAGE IN CHILDREN Keep preparations with aspirin out of reach of children. Equagesic *-M is not recommended for adients 12 years of age and under. PRECAUTIONS: ASPIRIN Sallcylates an-

fagonize uricosuric activity of probene-cial and suffinpyrazone. Salicylotes are reported to enhance hypoglycemic et-tect of sulforiyurea antidiabetics. MEPROBAMATE: Use lowest effective dose, parficularly in eldetly and/or debil-ifoted, to preclude over-sedation. Me-probamate is metabolized in the liver and excreted by the kildney; 10 avoid ex-cess accumulation exercise acufron in its use in patients with compromised liver or kidney function. Meprobamate occo-sionally may precipitate seizures in epi-leptic patients. It should be prescribed coullously and in small quantifies to pa-tients with sulcidal tendencies.

sionally may precipitate seizures in epileptic patients. It should be prescribed
caullously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS: ASPIRIN May
cause epigastria discomfort, nauser,
and waiting Hypersensitivity reactions,
edema, purpura, asthma, and anophylaxis may rately occur. Patients receiving
large doses of salicytates may develop
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MEPROBAMATE: CNS: Drowslness,
ataxia, dizzlness, slurred speech, head
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bullous dermatitis have occurred.
HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, oplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic

purpura.
OTHER Exacerbation of porphyric

DOSAGE AND ADMINISTRATION. Usual dose is one or two toblets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recom-mended for potients 12 years of age and

mended for potients 12 years of age and under OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomilting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both ospilin and meprobomate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treofment should include management of hyperthermia, specific parenteral electrolity therapy for ketacidosis and dehydrotion, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia whole-blood occurs, usually requires whole-blood uence a nemorrhagic manifestations due to hyporathrombinemia which, if it occurs, susually requires whole-blood fransfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethogy, stuppor, ataxia, coma, shock, vasamafor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature of the control of the control

BLOOD LEVELS:
0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.
3-10 mg percent usually corresponds to

findings of mild-to-moderate symptoms of overdosage, such as stupor or light

of overlosage, such as slaper or me...
coma
10-20 mg percent usually corresponds to
deeper coma, requiring more intensive
treatment. Some fatalities occur.
At levels greater than 20 mg percent,
more fatalities than survivols can be

more talcilities than survivols can be expected.
Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood of tissue level) cannot be used as a prognostic indicator.

(or of a relatively law blood of Issue ievel) cannot be used as a prognostic Indicator. Indicator. In case of excessive doses, sleep ensues in case of excessive doses, sleep ensues to be a prognostic prognosti

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S President's Page

About Our New President

JOSEPH N. HAMM was born in Winner, South Dakota and later moved to Rapid City where he graduated from Cathedral High School. He earned a Bachelor of Science degree in medicine at Creighton University in Omaha, Nebraska following which he was given a fellowship in Anatomy at George Washington University School of Medicine where he subsequently graduated with a degree of Doctor of Medicine. He completed one year of rotating internship at the University Hospital.

A surgical residency was curtailed by military service in the United States Navy during World War II. After a brief duty with a construction battalion he was assigned to the Naval Hospital ship, the USS Samaritan, for duty in the Pacific theater until the end of hostilities. He was discharged from military duty at the US Naval Hospital in Portsmouth, Vir-

ginia. He spent an additional one and one-half years in surgical training at the Veterans Administration Hospital in Washington, D. C. and a preceptorship in Internal Medicine.

He has been a member of the South Dakota State Medical Association and the American Medical Association since he opened an office for general practice in Sturgis, South Dakota in 1947. He joined the faculty of the School of Medicine of the University of South Dakota in 1975, and is presently situated in the West River offices of the School of Medicine in Rapid City.

He married Dorothy M. Eddington, formerly of Faith, South Dakota. They have two sons, Joseph N. Jr. in retail business in Rapid City, and Dr. Robert M. a radiologist in Loveland, Colorado.

South Dakota Society Of Pathologists

Officers for 1982-83

Jerry L. Simmons, M.D., President Thomas E. Henry, M.D., Vice President Beth L. Johnson, M.D., Secretary-Treasurer



S President's Page



May I first express my sincere gratitude to the members of this Association for selecting me to serve as your president for the coming year. I look forward to working with our splendid staff. I will need the support of each of you, the members, and I recognize the challenge of performing the duties of the office as well as did Dr. Durward Lang, and the others who have preceded him.

By the time that this is read the House of Dele-

gates will have adjourned their annual meeting. The Commissioners and the Council will convene regularly to represent you in the efforts of the Association, dedicated to providing excellent health care for our people. Bureaucratic interference will continue. It threatens to increase. It has been said that perpetual vigilance is the price of liberty. We will watch and continue to serve as we have for the first of our hundred and one years.

Joseph N. Hamm, M.D., President South Dakota State Medical Association

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S Clinicopathologic Conference

Sixty-One Year Old Caucasian Man with Rheumatoid Arthritis and Renal Failure

Daniel J. Heinemann, M.D.* Walter Drymalski, M.D.† Richard Jaqua, M.D.‡ Discussers

John F. Barlow, M.D.§
Editor

CASE #974 723

This 61 year old Caucasian male was admitted to Sioux Valley Hospital for evaluation of renal failure.

The patient had had known rheumatoid arthritis for approximately 20 years. He had been treated with many medications including non-steroid inflammatory agents such as phenylbutazone and indomethacin intermittently for years. The patient had been taking steroids for 10 to 12 years and was presently taking prednisone, 5 mgs. every other day. He had never taken penicillamine, hydroxychloroquin or gold. He had been given a course of cyclophosphamide but this was stopped after he lost his hair. The patient had had multiple operative procedures on joints including hip and knee joint replacement in the preceding several years. The knee joint replacement had to be revised. He also had a diagnosis of a duodenal ulcer without significant hemorrhage and sigmoid diverticulosis. During his previous admission, the most recent being 2½ years previously, the blood urea nitrogen and creatinine had been within normal limits and there had been no protein in the urine. Over the past eight months, the patient had noted increasing edema and had not felt well. Five months prior to admission, a serum creatinine was 1.5 mg/dl, a blood urea nitrogen was reported as elevated and he had 2+ proteinuria. Three months prior to admission, serum creatinine determinations were 2.5 and 3.6 mg/dl; blood urea nitrogen was 50 mg/dl and creatinine clearance was 35 ml/min. The patient had been given digitalis and diuretic therapy because of increasing edema and had developed an episode of digitalis toxicity which was relieved by discontinuing the medication. Two months prior to admission, the patient developed pneumonia and was admitted to another hospital. A creatinine was 6.0 mg/dl and blood urea nitrogen was markedly elevated. A low serum albumin, increased serum phosphate, markedly elevated rheumatoid arthritis titer (1 to 1200) and erythrocyte sedimentation of 145 mm/hr were reported during that admission. The fluorescent antinuclear antibody test (FANA) was negative.

There was no past history of urinary stone, hematuria or

PHYSICAL EXAMINATION: Temperature 98°F; pulse 76/ min. and reg; respirations 18/min. and reg; blood pressure 120 systolic and 62 diastolic. The patient was a very thin gentleman with parchment-like skin and numerous bruises. There were bilateral cataracts. The patient had a hearing aid on the left side. There was no other abnormality to the head and neck. The carotid pulses were 4+ and equal. The chest was clear to auscultation and percussion. The heart was not enlarged. There were no murmurs, rubs or gallops. There was a split S1. Examination of the abdomen revealed no palpable organs or masses but there was slight tenderness without spasm in the right upper quadrant. The genitalia were normal. There was 3+ pitting edema below the knees. There were marked deformities of the joints involving the wrists, elbows, knees and ankles. There was a hammer-toe deformity of the left second toe. There was decreased motion of the cervical spine. The pulses were 4+ and equal bilaterally. Rectal examination was negative.

LABORATORY DATA: Urinalysis showed 4+ proteinuria. There were no significant abnormalities of the sediment. Hemoglobin was 9.7 gm/dl; hematocrit 31 vol/dl with

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[§]Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

normal red cell indices; total leukocyte count 8,400/mm3 $(8.4 \times 10^9/L)$ with 87% segmented neutrophils, 1% neutrophilic bands, 12% lymphocytes; platelet count 710,000/ mm3 (710 \times 10 $^{9}/L$). The red cells showed slight anisocytosis and polychromasia on smear. An erythrocyte sedimentation rate was 140 mm/hr. Lactic dehydrogenase (LDH) was 272 IU/L (normal 0-270 IU/L); calcium 7.3 mg/dl (normal 8.4-10.7 mg/dl); phosphate 5.3 mg/dl (normal 2.1-4.5 ml/dl); blood urea nitrogen 70 mgs/dl, creatinine 6.4 mg/dl, cholesterol 604 mg/dl. The alkaline phosphatase, aspartate aminotransferase (AST); total bilirubin, glucose, sodium, potassium were within normal limits. A chloride was 113 meq/L, total CO2 11 meq/L, total protein 5.0 gm/dl, with 1.8 gm/dl albumin; 0.4 gm/dl alpha I globulin; 1.2 mg/dl alpha II globulin; 1.0 gm/dl betaglobulin; 0.6 gm/dl gammaglobulin; arterial blood gases - pH 7.41, PCO2 21 torr; CO2 content 13 meg/L, PO2 93 torr, O2 saturation 96% on room air. Urine protein 10.6 gm/24 hours; creatinine clearance 6 ml/ min. Urine calcium 7 mg/24 hours (normal 50-300 mg/24 hours); urine uric acid 335 mm/24 hours (normal 600-800 mg/24 hours). Prothrombin time and partial thromboplastin time were within normal limits. A test for serum cryoglobulins was negative. Test for rheumatoid factor was 240 IU/ml (markedly elevated); FANA test, negative; serum complement 41 units (41 to 90 units); urine and serum immunoelectrophoresis - no diagnostic pattern but consistent with renal disease. Tests for serum immune complexes was negative. An electrocardiogram showed a sinus rhythm with non-specific changes. A bilateral renal ultrasound showed calculi in the right kidney. There was no evidence of hydronephrosis. There were a few cysts in the right kidney; the right renal length was 8.8 cms and the left renal length 9.8 cm. X-rays of the skeletal system showed extensive arthritis of the cervical spine and extensive rheumatoid involvement of the left elbow and wrist. There was invagination of a large, irregular odontoid process. There was dystrophic calcification in the left elbow with an accompanying effusion. An open left renal biopsy was performed.

DR. HEINEMANN: I thought that I would start the discussion of this case by talking about the evaluation of renal failure. Renal failure is the term applied to a general loss of renal function, and the accumulation of substances like phosphate, sulfate and potassium. All of these compounds are dependent on normal glomerular filtration for their removal from the body. Common symptoms of uremia, the clinical syndrome of renal failure, are malaise, weakness, nausea and vomiting. Pericarditis, twitching and convulsions are more serious symptoms. In chronic renal failure osteodystrophy and neuropathies often become evident.

Commonly, renal failure is categorized as either pre-renal, renal or post-renal. It is important for the clinician to distinguish these types so as to best help his patient. One author used an extensive breakdown and uses the mnemonic A-E-I-O-U, Corporation Vice President Hates Dogs Mating. These letters stand for various entities which may be involved in the renal failure A = acute, E = electrolyte abnormalities, I = infection, O = obstruction, U = uric acid, C = collagen disease, V = vascular, P = prerenal and pregnancy, H = hypertension, D = drugs and M = miscellaneous.

Based on the above possibilities the workup should include:

History: The physician should search for predisposing disease and possible drug ingestion.

Physical Examination: The examination should include blood pressure determination, examination of the fundi for changes of hypertension, heart for presence of murmurs and enlargement, as well as search for an enlarged spleen, renal tenderness, enlarged prostate or enlarged bladder.

Laboratory Studies: Complete urinalysis should be done as well as serum sodium, potassium, calcium, blood urea nitrogen, creatinine, uric acid and creatinine kinase (CK). Urine sodium and creatinine are important. One should also obtain blood and urine cultures if indicated. The initial workup should also contain a single catheterization of the bladder to rule out bladder neck obstruction.

Pertinent radiologic studies include KUB (to determine renal size), intravenous pyelogram with tomography and retrograde studies if obstruction is a possibility. Renal ultrasound or computer tomographic scan may be albe to provide valuable information. Finally, if vascular obstruction is a possibility, the renal arteriogram or renal venogram should be considered. If diagnosis is still in doubt either needle or open biopsy of kidney should be performed.

Acute renal failure is really a clinical not a pathologic entity because no permanent damage involves the kidneys and gradual spontaneous recovery ensues over a period of days to weeks.

Acute oliguric renal failure is seen in the following conditions: 1) episodes of renal ischemia, including those secondary to hypotension, 2) exposure to nephrotoxic agents, 3) release into circulation of heme pigments, mainly hemoglobin and myoglobin. It is not the pigment but associated clinical conditions related to the release of these pigments which produces renal failure.

The diagnosis of acute renal failure is primarily one of exclusion of the various possibilities listed above but consideration may also include the acute onset of chronic renal failure.

The prognosis for patients is poor. About 50% of patients die with infection being the leading cause of death. Survivors, however, usually attain normal or near normal renal function. The majority of patients with acute azotemia have oliguria, since anuria usually points to obstruction or very severe parenchymal disease. Oliguria is less than 400 cc/urine/day, anuria less than 50 cc/day. One of the best parameters to aid in the diagnosis of the cause of acute renal failure is the fractional excretion of sodium. The formula and significant values are as follows:

$$FE_{Na} = \frac{(U/P) \text{ ng}}{(U/P) \text{ cr}} \times 100$$

	Urine Osm	Urine Na	FE Na
pre renal	plasma	20	1
post renal	Isotonic	variable	2
renal	Isotonic	20	2

Using this formula and the values obtained, the physician is able to get good indication if the renal failure is caused by low blood flow to the kidneys, obstruction or intrinsic renal disease.

Chronic renal failure is the term used to describe a marked chronic decrease in glomerular filtration that is ordinarily irreversible. There is usually a loss of both glomerular and tubular functions. It is the loss of glomerular functions that is responsible for the classic symptoms of uremia. Commonly patients complain of weight loss, nausea, bad taste in the mouth and generalized malaise.

The causes of chronic renal failure are numerous and include any disease that results in destruction of renal tissue. Common examples are chronic glomerulonephritis, tubulointerstitial disease, diabetic nephropathy and polycystic kidney disease.

Diagnosis is established by history of known renal disease, presence of symptoms and laboratory values; and, most importantly, by objective evidence of loss of kidney mass. One must rule out a reversible cause of renal failure.

Renal biopsy may not provide much information to physicians if the kidneys are small. If however, kidneys are not small, biopsy may provide valuable information e.g. amyloidosis, sarcoidosis, or infiltration by lymphoproliferative disorder. Knowledge of underlying disease may aid in treatment selection.

This patient has many of the classic laboratory abnormalities of renal failure and nephrotic syndrome — proteinuria, normochromic normocytic anemia, hypocalcemia, hyperphosphatemia, elevated serum creatinine, blood urea nitrogen, cholesterol, hypoproteinemia, depressed creatinine clearance, urine calcium and uric acid.

This patient also has significant rheumatoid arthritis and has had treatment with medications which could cause acute renal failure. At the time of onset of his renal failure, he was taking no medications which one could implicate as the causative agent. His renal failure had an acute onset with gradual insidious progression. This leads me to believe that the cause of his renal failure is related to some complication of his arthritis and may or may not be reversible. Hopefully, the kidney biopsy will give us the information we need. Amyloidosis, chronic glomerulonephritis or interstitial nephritis from anti-inflammatory agents are possibilities.

Dr. Heinemann's Diagnosis Renal Failure From Possible Amyloidosis, Glomerulonephritis or Interstitial Nephritis

DR. JAQUA: Examination of the renal biopsy revealed severe parenchymal damage involving glomeruli, vessels and interstitium. The biopsy shows severe interstitial fibrosis with tubular atrophy, totally and partially sclerosed glomeruli with a relatively homogenous eosinophilic material most prominent in mesangial or axial regions as well as in peripheral capillary location (Fig. 1). There is minimal proliferative component involved in the glomerular damage and the periodic acid-Schiff (PAS) stains show that this material has less PAS reactivity than basement membranes or the proteinaceous casts present. The trichrome stains revealed the extensiveness of the tubulointerstitial disease with considerable interstitial and periglomerular fibrosis and a faint staining of the nodular increase in mesangial material. The silver stains which sharply outline the basement membrane areas show obscuring of the basement membrane in areas and one can observe in the lower portion of some glomeruli spicular-like outgrowths of silver staining material on the epithelial side of the capillary. This has a superficial resemblance to the sawtoothed appearance of membranous glomerulopathy.



Figure 1
Center of picture shows two glomeruli markedly altered by deposition of amyloid.

However, it has been described as a highly suggestive feature of renal amyloid accumulation by Nolting and Campbell.⁴ The immunofluorescent studies yielded some rather surprising results. There was rather intense staining of the glomerular and vascular deposits with fluorescent conjugated antibodies to immunoglobulin G and complement components

C-3, C-4 and C-1q and both kappa and lambda light chains. There is no staining of the deposits with the anti-IgM and anti-IgA antisera or the fibrinogen or albumin reagents suggesting that this is not a nonspecific protein trapping phenomenon. This is an unusual finding in amyloid disease, especially of the secondary type which one would expect in this case. However, as has been pointed out by Jerath et al,⁵ a great variety of different patterns of immunoglobulin and complement localization have been described in amyloid and although they have not been successfully explained, the deposition does seem to involve specific components as in the present case. Many of the cases, however, show no immunoglobulin or complement component localization at all. There are a variety of confirmatory methods when one suspects amyloid disease of the kidney. Probably the most commonly used and well accepted histochemical method is the Congo red stain combined with polarizing microscopy. On bright field microscopy one can see the deposits do seem to take up the Congo red stain and under the polarized microscope, one does see the apple green type of birefringence which is highly characteristic for but not entirely specific for the amyloid protein. Another technique that can be used with the Congo red stain is fluorescent microscopy in which the amyloid will show a dark orange fluorescence. A more recent histochemical technique suggested for amyloid demonstration involves the toluidine blue stain which shows some degree of metachomasia represented by reddish purple staining. Under polarized light amyloid will give a bright red birefringence.

The most sensitive and specific method we have at present to confirm the presence of amyloid is electron microscopy which is routinely carried out on our renal biopsies. One can see in this slide portion of a capillary with the fibrillar material in subendothelial intramembranous and subepithelial locations. (Fig. 2) This electron micrograph also shows a portion of a glomerular epithelial cell or podocyte showing some villous transformation and projecting into its cytoplasm from the capillary wall are bundles of the typical beaded 10nm amyloid fibrils. It is this type of spicular projection which is seen on the silver stains in light microscopy.

Amyloidosis is a very poorly understood protein deposition disease which has been classified in many different ways. There are several different major protein components in the different clinical types. Primary amyloidosis, multiple myeloma, localized and endocrine amyloid have a common major protein component which consists of portions of the variable region of light chains of immunoglobulins (AL amyloid). The other major category of amyloidosis is secondary amyloidosis which is associ-



Figure 2 Electron Micrograph showing typical fibrillary pattern of amyloid in glomerular capillary wall.

ated with chronic inflammatory conditions such as chronic infection and rheumatoid arthritis as in the present case. Secondary cases are associated with deposition of morphologically identical fibers which contain a portion of an acute phase reactant normally seen in serum called protein A or SA, the function of which is not understood. At the present time, specific identification of the protein type in amyloidosis is neither practical nor clinically useful. However, there are some simple methods described which allow one to differentiate the AA amyloid type from the AL amyloid type, namely the permanganate digestion technique. It has been found that permanganate treatment will in some way inhibit the uptake of Congo red in the type AA amyloid seen in secondary amyloidosis where the AL type of amyloid seen in primary amyloidosis, multiple myeloma, and localized amyloid will be unaffected by this treatment. Wright and Calkins⁶ in a recent review of the amyloid syndromes in medicine found that the permanganate technique was extremely useful in separating primary from secondary cases. The fluorescent Congo red stain in the present case demonstrated complete inhibition of staining by the permanganate treatment confirming that this is indeed the AA type of amyloid as one would expect in chronic rheumatoid arthritis. In this same article the above authors confirm that the most common causes of secondary amyloidosis are chronic pyogenic infections and rheumatoid arthritis.

FINAL ANATOMIC DIAGNOSIS AMYLOIDOSIS OF KIDNEY, SECONDARY TYPE

DR. HEINEMAN: How often does amyloidosis

occur in rheumatoid arthritis?

DR. DRYMALSKI: Clinically significant amyloidosis occurs commonly in juvenile rheumatoid arthritis, up to 20% in some series but is much less common in adults. This is only the second or third case that I have seen. One must remember that rheumatoid arthritis can be active or inactive. The indication for treatment depends on the activity of the disease. However, one must also assess the activity of the disease in joints. If there is already advanced destruction, treatment may be of little help. Methods of clinical assessment of the activity of the disease include clinical evaluation of redness and swelling of the joints, the erythrocyte sedimentation rate, platelet count and aspiration of the joint. In the latter case, if there are over 5000 cells/ mm3, activity is probably present. Patients with active rheumatoid arthritis also develop severe anemia. In this particular case, the anemia could well be due to renal failure. Since the anemia is often normochromic normocytic in both rheumatoid arthritis and renal failure, the red cell indices are not of much help. The level of the rheumatoid factor is of some help in evaluation of the activity of rheumatoid disease. We did treat this patient by raising the steroid dosage because of the significant amyloidosis. We also are treating the patient with azathioprine. We are monitoring the therapy by the erythrocyte sedimentation rate.

It is interesting that this patient had not received gold, penicillamine, or hydroxychloroquin. I am not sure why this is the case. He did receive cyclophosphamide but under a physician's direction.

The causes of renal disease in rheumatoid arthritis are limited. Rheumatoid vasculitis or interstitial nephritis from the anti-inflammatory agents could cause renal failure as well as amyloidosis, which is, of course, the first thought. If the patient had Sjorgren's syndrome, renal tubular acidosis could develop.

DR. MARSCHKE*: Do you think the amyloidosis is reversible in this patient?

DR. DRYMALSKI: I am not sure if it is reversible, but I think we can limit the progression of the disease.

DR. MARSCHKE: Should the patient receive a renal transplant?

DR. DRYMALSKI: Yes, but amyloidosis might develop in the transplanted kidney. However, the patient's immune system would be suppressed after the transplantation.

DR. JAQUA: Yes, recurrence of amyloid in the renal transplant has been reported.

DR. MARSCHKE: Would a biopsy of some other tissue in this patient such as the rectum or gingiva have been as rewarding in making the diagnosis? DR. DRYMALSKI: It might have made the diagnosis of amyloidosis, but wouldn't necessarily tell you what was the cause of the renal disease.

DR. JAQUA: Rectal biopsies are very effective in making the diagnosis but I agree that in this case the nature of the renal disease had to be determined.

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^{*}Oncologist, Central Plains Clinic and Sioux Valley Hospital; Assistant Clinical Professor of Medicine, School of Medicine, University of South Dakota, Sioux Falls, SD.

14th ANNUAL

BLACK HILLS SUMMER SEMINAR

ON FORENSIC MEDICINE, AGRICULTURAL AND OCCUPATIONAL MEDICINE, PSYCHIATRY

AUGUST 11, 12, 13, 1983

HOWARD JOHNSON MOTOR LODGE, RAPID CITY, SOUTH DAKOTA

Hosted by South Dakota Chapter of the American Academy of Family Physicians
This program has been reviewed and is acceptable for 16 prescribed hours by the American Academy of Family Physicians and 16 hours Category 1 AMA Physician Recognition Award.

WEDNES	SDAY, AUGUST 10, 1983	8:40-9:20 a.m.	A Look at Occupational Medicine
4:00 p.m.	SDAFP Board of Directors Meeting	9:25-9:45 a.m. 9:50-10:30 a.m.	Edward H. Peters, M.D. Coffee, Conversation, Consultation Anhydrous Ammonia, Sulfides, Nitrates
THURSI	DAY, AUGUST 11, 1983	10:35-11:15 a.m.	Donald P. Morgan, M.D., Ph.D. Zoonoses Update — Bovine
6:30-7:00 a.m.	Registration		Leukemia/Human Leukemia Relationships
7:00-7:10 a.m.	Complimentary continental breakfast Welcome Herbert Saloum, M.D.	11:20-11:45 a.m.	Kelley J. Donham. D.V.M. Question and Answer Session
7:10-7:40 a.m.	History of Medical-Legal Death Investigation	12:00-2:00 p.m.	Hosted buffet luncheon for registrants Informal panel with speakers SDAFP annual business meeting and
7:45-8:15 a.m.	Bradley B. Randall, M.D. Pathology of Child Abuse Thomas E. Henry, M.D.		election of officers
8:20-8:50 a.m.	Laboratory Investigation of Rape Bradley B. Randall, M.D.	REMAINDER OF AFTERNOON AND EVENING FREE	
8:55-9:15 a.m. 9:20-9:50 a.m.	Coffee, Conversation, Consultation Blunt Force Trauma		
9:55-10:25 a.m.	Thomas E. Henry, M.D. Gunshot Wounds	SATURDAY, AUGUST 13, 1983	
	Bradley B. Randall, M.D.	6:30-7:00 a.m.	Registration
10:30-11:00 a.m.	Sudden, Unexpected Deaths Thomas E. Henry, M.D.	7:10-7:50 a.m.	Complimentary continental breakfast Depression, Part I — Diagnosis
11:05-11:20 a.m. 11:30-1:15 p.m.	Question and Answer Session Hosted luncheon for registrants and	7:55-8:35 a.m.	Joseph H. Talley, M.D. Evaluation of Risk Factors for Teenage
* 20	spouses Gerald R. Gehringer, M.D., AAFP President-Speaker	8:40-9:20 a.m.	Pregnancy Bill Arbes, Ph.D. Depression, Part II — Therapy
1:30 p.m.	Legislative Committee, SDAFP Education Committee, SDAFP	9:25-9:45 a.m. 9:50-10:30 a.m.	Joseph H. Talley, M.D. Coffee, Conversation, Consultation Counseling Parents of Sexually Active
REMAINDER OF	AFTERNOON AND EVENING FREE		Adolescents Bill Arbes, Ph.D.
		10:35-11:15 a.m.	Anxiety Disorders (Panic Disorders & Generalized Anxiety Disorders)
FRIDAY, AUGUST 12, 1983		11:20-11-50 a.m.	Joseph H. Talley, M.D. Evaluation of Impotence for Penile
6:30-7:00 a.m.	Registration		Prosthesis Implant Bill Arbes, Ph.D.
7:10-7:50 a.m.	Complimentary continental breakfast Toxicology of Agricultural Chemicals Donald P. Morgan, M.D., Ph.D.	11:15-12:30 p.m.	Question and Answer Session
7.55-8.35 a m	Agricultural Passington, Diseases		

MAKE PLANS TO ATTEND NOW, WRITE: BLACK HILLS SUMMER SEMINAR c/o South Dakota Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104

SEMINAR CLOSES

Agricultural Respiratory Diseases —

Livestock Confinement Kelley J. Donham, D.V.M.

7:55-8:35 a.m.

S Chapter News





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

Nominating Committee Report

The SDAFP Nominating Committee submits the following slate for consideration at the Annual Meeting to be held in Rapid City, August 12, during the Black Hills Summer Seminar:

President-Elect:

Charles L. Swanson,

M.D., Pierre

Vice President:

Richard W. Honke, II,

M.D., Wagner

R. Curtis Jahraus, M.D.,

Pierre

(one to be elected)

Secretary/Treasurer: Loren H. Amundson.

M.D., Sioux Falls

Delegate: Bruce C. Lushbough,

M.D., Brookings

Alternate Delegate: Ray G. Nemer, M.D.,

Gregory

Nominations may be made from the floor at the annual meeting.

State Officers Conference

Your SDAFP Board of Directors all attended the State Officers Conference in Kansas City recently, hearing talks by Senator Robert Dole and retired UCLA basketball coach, Mr. John Wooden.

A Board of Directors meeting was also held. Others attending were: Delegate, R. W. Friess, M.D. and State Education Chairman, Bruce Lushbough, M.D.

RRC to Residency Program

The Residency Review Committee for Family Practice visited and reviewed the Sioux Falls program in April. A report of this visit by the accrediting body is expected in July.

DAP at USDSM

The Department of Family Medicine at USD School of Medicine had a Department Assistance Program (DAP) consultation recently. This was conducted by Thomas A. Nicholas, M.D., Chairman of Family Medicine at the University of Missouri, Kansas City.

ABFP Board of Directors

Loren H. Amundson, M.D., Secretary/Treasurer of SDAFP, was elected to a five year term on the Board of Directors of the American Board of Family Practice.

The ABFP is the specialty board that examines and re-examines diplomate candidates for specialty status in family practice.

PATIENT EDUCATION TIPS

Even a Little High Blood Pressure Is Too Much

Blood pressures at the low end of the high blood pressure scale are sometimes called mild. Yet lower level elevations, if untreated, can still lead to serious problems like heart disease, stroke, and kidney disease.

Fortunately, treatment can make a big difference in reducing potential problems for people in this group. It can also prevent mild high blood pressure from going to higher ranges where serious complications are even more likely.

You can encourage your patients to remember that even a little high blood pressure is too much. Patients may ask about steps they can take to keep their blood pressure problem under control.

FOR SALE

1850 square foot modern nicely located medical office. Fully equipped. 1 to 2 man practice. Four complete examination rooms. Complete GE x-ray and developing equipment. Medically deprived community with 3 other full time practitioners. 42 miles east of Billings, Montana. Modern 20-bed hospital serves an estimated area of 10,000 people. Available in July 1983. Seller will finance equipment and building. Well established practice that has been extremely busy. Located near the famous Big Horn River with ideal fishing, excellent hunting, skiing in nearby mountains. For further information con-

Daniel J. Gebhardt, M.D. 203 West 4th Hardin, MT 59034 Phone: (406) 665-3400 Office

(406) 665-3339 Home (evenings)

Family Physicians

Unique opportunity for BC/BE Family Practice Physician to join prepaid group practice in Kansas City. To staff and develop a family practice facility 15 minutes from established multi-specialty group practice. Facility will include laboratory, X-ray, and pharmacy. Attractive salary structure and liberal fringes. Starting salary based on experience. Recruitment and relocation expenses covered.

Send CV to:

Michael R. Soper, M.D. 6801 E. 117th Street Kansas City, Missouri 64134 or call (816) 765-6200

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Bactrim DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

which follows: Indications and Usage: For the treatment of urinary tract Infections due to susceptible strains of the following organisms: Escherichia coli, Klebsiella-Enterobacter, Profeus wirabills, Profeus wilgaris, Profeus morganii. It is recommended that Initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. Note: The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute othis media in children due to susceptible strains of Haemophilus

Influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not Indicated for prophylactic or prolonged administration in otitis media at any

age.

For acute exacerbations of chronic bronchitis in adults due to susceptible

age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of Haemophilus influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over a single antimicrobial agent. For enteritis due to susceptible strains of Shigelia flexneri and Shigelia sonnel when antibacterial therapy is indicated. Also for the treatment of documented Pneumocystis carinii pneumonitis. Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with timethopmin is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, lever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin, reassess coagulation time when administering Bactrim to these patients. Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Allergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruntus, exfoliative dermatitis, anaphylactoid reactions, penorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. Gastrointestinal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous coli-

allergic myocarditis. Gastrointestinal reactions: Glossilis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. CNS reactions: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. Miscellaneous reactions: Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies. Dosage: Not recommended for Intants less than two months of age. URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 morks trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two

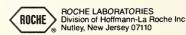
media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis. For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min, Its creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is between 15 mg/lgip. below 15 ml/mir

below 15 mil/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or
4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:
Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per
24 hours in equal doses every 6 hours for 14 days. See complete product information
for suggested children's dosage table.

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and
800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100;
Prescription Paks of 20. Tablets, each containing 80 mg trimethoprim and 400 mg
sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription
Paks of 40. Pediatric Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz
(1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole
per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

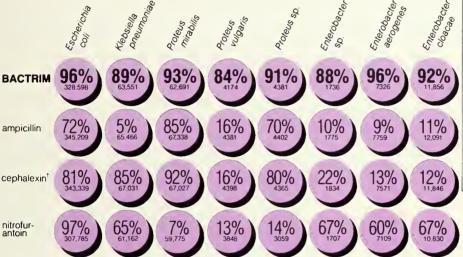




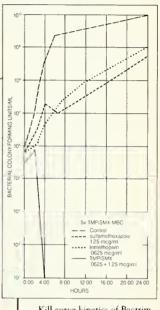
Bactericidal activity

with minimal resistance

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials



[†]Analogous to cephalothin, the primary antibiotic disc used in testing. Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82. Numbers under percentages refer to the projected number of isolates tested. RAPID IN VITRO DESTRUCTION OF E. COLL*



Kill curve kinetics of Bactrim and its individual components against E. coli in vitro. 1

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*. ^{1,2} and on clinical isolates of *E. coli*, *Klebsiella-Enterobacter*, *Proteus mirabilis* and *Morganella morganii*³—the most common causative organisms of urinary tract infections. ⁴ More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms. ⁵ In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy. ⁶⁻¹¹

Resistance to Bactrim develops more slowly than to either of its components alone in vitro.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains. ^{5,12} Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency. Initial episodes of

uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. for recurrent urinary tract infections

*In vitro data do not necessarily predict clinical results.

Motrin[®] ibuprofen, Upjohn 600 mg Tablets





More convenient for your patients

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S Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday, April 22, 1983, at the Ramada Inn, Sioux Falls, South Dakota. Following are the items of business transacted at this meeting.

- 1. PART B MEDICARE. The Council directed that the State Medical Association continue to see representation on the medical claims review committee of North Dakota Blue Shield, South Dakota's Part B Medicare carrier.
- 2. COST CONTAINMENT REPORT. The Council received a report from the Cost Containment Subcommittee of the Commission on Medical Service and voted to defer action on this report to give the district medical societies an opportunity to review the report and bring recommendations back to the Council at a future meeting; at which time the Council will give specific direction to the Commission.
- 3. NURSERY SUPERVISORY COMMITTEE REPORT. The Council received the proposed guidelines for newborn and obstetrical care and voted to defer action to give each district medical society an opportunity to review this report and bring recommendations back to the Council.
- 4. GOVERNOR'S REQUEST FOR ASSIST-ANCE REGARDING MEDICAID COSTS. The Governor requested the advice of the State Medical Association in three specific areas concerning Medicaid costs. The Council voted to establish an ad hoc committee to define essential medical services and to establish methods for the care of the terminally ill patient and to provide these recommendations to the Governor. With regard to Indian health care problems, the third area addressed by the Governor, the Council referred oversight of this to the Liaison Committee inasmuch as both the Health Department and the Medical School are currently addressing this problem.
- HONORARY MEMBERSHIP. The Council voted Dr. Charles Stern and Dr. Kendall Burns honorary life membership in the State Medical Association.
- 6. ENDOWMENT BOARD OF DIRECTORS. The Council reappointed the following to serve a one year term on the South Dakota Medical School Endowment Board of Directors: G. E. Tracy, M.D., Bruce Lushbough, M.D., T. H. Sattler, M.D., Warren Jones, M.D., Robert

- Giebink, M.D., Joseph Hamm, M.D. and Bruce Allen, M.D.
- PROPOSED REVISIONS TO JCAH ACCRED-ITATION MANUAL FOR HOSPITALS. The Council supported the AMA's position that the wording "medical staff" be retained in the proposed revisions of the JCAH standards rather than "organized staff."
- 8. VACANCY ON BOARD OF MEDICAL AND OSTEOPATHIC EXAMINERS. The Council recommended Dennis Johnson, M.D., Sioux Falls, for the Governor's consideration for appointment to the State Board of Medical and Osteopathic Examiners.



S

This Is Your Medical Association

John F. Barlow, M.D., Sioux Falls pathologist, was honored recently by the USD School of Medicine Department of Pathology, the Laboratory of Clinical Medicine and Sioux Valley Hospital for authoring the Clinicopathological Conferences, which have appeared monthly in the South Dakota Journal of Medicine, for the past seventeen years. Austin Vickery, M.D., Professor of Pathology at Harvard Medical School and Massachusetts General Hospital, along with many of Dr. Barlow's colleagues, former students, interns and residents were present at this special reception.

Governor Janklow has appointed **Dr. Michael Rost,** Sioux Falls, to a six year term on the state Board of Regents. Dr. Rost is chief anesthesiologist at McKennan Hospital and also president of the State Board of Medical and Osteopathic Examiners.

Yankton doctor, **R. I. Porter**, has completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

Terry Lang, M.D. was elected to the Fellowship in the American Academy of Pediatrics. Dr. Lang is an associate professor of pediatrics at the USD Medical School in Sioux Falls.

Three Huron physicians, **Drs. Paul Tschetter**, **Ted Hohm**, and **Paul Hohm**, received the Service to Mankind Award in recognition of community achievement by the Huron Sertoma Club, at their annual banquet.

A. R. Scheffel, M.D. and D. M. Patterson, M.D., both of Redfield, were recently honored at the Chamber of Commerce annual meeting and banquet for their years of service to the Redfield Community. The two doctors have a total of nearly 60 years of medical service between them.

Dennis C. Stevens, M.D., Sioux Falls, was appointed Executive Chairman of the March of Dimes WalkAmerica. Dr. Stevens is a neonatologist at Sioux Valley Hospital.

Isabel Sattler, Yackton, has been named South Dakota Mother of the Year for 1983. Isabel and her husband **Dr. Theodore Sattler** have two daughters and one grandson. She is a South Dakota native, born in Beresford. She is active in: Greater Yankton United Way, Contact Center, Questers, Heritage House, Chapter K, PEO, AAUW, Leage of Women Voters and is the secretary of the Yankton County Historical Society.

Ray E. Lemley, M.D., Rapid City, died March 18, 1983, at the age of 80. He was born June 13, 1902 in Rapid City. He graduated from Macalester College in St. Paul, Minn. in 1923 and the U. of Minnesota School of Medicine in 1929. In 1936, he was the first doctor to move from private offices in Rapid City to establish a clinical practice.

He studied at the U. of Vienna in 1937 during Hitler's occupation of Austria and he smuggled out some of the only existing pictures of that occupation. He also studied techniques in urological surgery at the U. of Hungary in Budapest in 1939 and while in Hungary he helped in the removal of many refugees before the Blitzkrieg.

Dr. Lemley is noted for his early research, in the 1940's, on the element selenium in animals and humans. His work was a basic step for some current cancer research.

After serving as a major, in the Army Air Corps during World War II, he co-founded Rapid City Medical Center. He retired in 1960.

Dr. Lemley is survived by his wife, Myretta; one son, William, Rapid City; a sister, Margaret Warren, Rapid City; and three grandchildren.

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David Cruz, M.D., family practitioner from St. Paul, Minnesota, has opened his practice in Corsica. Dr. Cruz is a native of the Philippines where he studied medicine and practiced for two years. He spent a year in Canada before moving to the United States in 1968 for training purposes. Since then he has lived in the Twin Cities area and owned a family practice clinic in Minneapolis for four years and then moved his practice to St. Paul.

Dr. Cruz and his wife have four children.

* * * *

Completing continuing education requirements to retain active membership in the American Academy of Family Physicians is J. C. Rodine, M.D., an Aberdeen physician.

* * * *

David Staub, M.D., of Sisseton, has completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

Dr. Ronald Price, an Armour physician, was presented a plaque for over 35 years of service, by the Armour Community Club. Dr. Price is a native of Ohio but grew up in Indiana. He came to South Dakota in 1947.

* * * *

D. A. Gehlhoff, M.D., Sioux Falls psychiatrist, has been renamed to the South Dakota Board of Charities and Correction by Governor Janklow. Dr. Gehloff is the director of mental health services at McKennan Hospital and a clinical associate professor at USD School of Medicine.

* * * *

Mitchell physician, **Dr. Robert Dappen**, has been notified that he passed his Internal Medicine Board Certification exam. He is now certified in Internal Medicine and is a member of the American Board of Internal Medicine.

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S Future Meetings

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- Eighth Annual Convention of the American College of International Physicians, Henry VIII Inn & Lodge, Bridgeton, MO, July 14-18. 1 hr. AMA Category 1 credits. Contact: Am. Coll. of International Phys., 3030 Lake Ave., Fort Wayne, IN 46805. Phone: (219) 424-7414.
- The Total Hip: Current Status, Hyatt Regency, Minneapolis, MN, July 28-30. Fee: \$350. 17 hrs. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- CME Caribbean Cruise/Conference on Legal-Medical Issues, Caribbean waters on board TSS FAIRWIND, July 27-Aug. 6. 24 hrs. AMA Category 1 credits. Contact: Linda Wynn, International Conf., 189 Lodge Ave., Ste. C, Huntington Station, NY 11746. Phone: (516) 549-0869.

August

- 1983 Black Hills Summer Seminar, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 11-13. Fee: \$100. 15 hrs. AAFP & AMA Category 1 credits. Contact: L. H. Amundson, M.D., 3001 S. Holly, Sioux Falls, SD 57105. Phone: (605) 335-5008.
- Annual Sixth District American College of Obstetricians and Gynecologists Meeting, Rushmore Plaza Civic Ctr., Rapid City, SD, Aug. 18-20. Contact: The Women's Clinic, 2805 Fifth St., Ste. #110, Rapid City, SD 57701. Phone: (605) 343-6550.
- The Fourth Biennial Leadbetter Symposium Urolithiasis: Biochemical, Metabolic, and Surgical Aspects, Willey Hall, U. of Minn., Minneapolis, MN, Aug. 18-20. Fee: \$350. 23 hrs. AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Regional Postgraduate Conference, Hyatt Regency, San Antonio, TX, Aug. 19-21. Fee: \$22.50 per hr. Contact: Jeanette Stone, Southern Med. Assoc., P. O. Box 2446, Birmingham, AL 35201. Phone: (205) 323-4400.
- CME Mediterranean Cruise/Conference on Legal-Medical Issues, Mediterranean waters on board MTS DANAE, Aug. 20-Sept. 3. 24 hrs. AMA Category I credits. Contact: Linda Wynn, International Conf., 189 Lodge Ave., Ste. C, Huntington Station, NY 11746. Phone: (516) 549-0869.

Medical Malpractice Seminar, The Homestead, Hot Springs, VA, Aug. 26-28. Fee: \$275. Contact: Jeanette Stone, Southern Med. Assoc., P. O. Box 2446, Birmingham, AL 35201. Phone: (205) 323-4400.

September

- Radiology/83: Special Imaging Including Computed Tomography, Ultrasound and Digital Angiography, Willey Hall, U. of Minn., Minneapolis, MN, Sept. 12-16. Fee: \$400. 28 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Occupational Health and Safety Institute, U. of Minn., Minneapolis, MN, Sept. 12-23. Graduate degree credit & CME credits. Fee: \$350 1st course; \$150 each course after. Contact: Ruth K. McIntyre, Dir. Con't. Ed., Midwest Ctr. for Occupational Health & Safety, 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Comprehensive Care of the Burn Patient, Hyatt Regency, Kansas City, MO, Sept. 16-17. Fee: \$225. 24 hrs. AMA Category 1 credits. Contact: Robert W. Gillespie, M.D., St. Elizabeth Comm. Hlth. Ctr., 555 S. 70th, Lincoln, NE 68510.
- Atlantic-Mediterranean Meeting on Gastroenterology, Mediterranean Cruise, Sept. 17-Oct. 7. Fee: \$1,640 includes passage, meals, and other extras. Contact: Mario Blanco Peres, Rua Goncalo, Cristovao, 116-3°, 4000 Porto, Portugal. Phone: 24294, 20933 or 31002. Telex: 26850 FRZDN-P.
- NIH Consensus Development Conference on the Treatment of Hypertriglyceridemia, Masur Aud., Warren Grant Magnuson Clinical Ctr., NIH, Bethesda, MD, Sept. 27-29. Contact: Mr. Peter Murphy, Prospect Assoc., 2115 E. Jefferson St., Ste. 401, Rockville, MD 20852. Phone: (301) 468-6555.

October

The Eighth Annual International Body Imaging Conference, Maui Surf Hotel, Maui, Hawaii, Oct. 8-16. Fee: \$395. 28 hrs. CME Category I credits. Contact: Conf. Secretary, Eighth Ann. International Body Imaging Conf., Dept. of Rad., West Park Hosp., 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (213) 340-0580.

February, 1984

Sportsmedicine Conference — Coincides with 1984 Winter Olympics, Dubrovnik, Yugoslavia — 20 min. from Winter Olympics. One and two week sessions throughout February, 1984. Fee: \$2,895 — 1 week; \$3,795 — 2 weeks (includes conf/tour pkg. and airfare). Conference only \$400 — 1 week; \$800 — 2 week. Contact: Robert Schaefer, School of Cont. Ed., Hahnemann Univ., Broad & Vine, Philadelphia, PA 19102.

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OUTH DAKOTA JOURNAL OF

Medicine

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Pulmonary Embolus with a Normal Ventilation Perfusion Lung Scan: Case Report

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NEXT MONTH

Transactions of the South Dakota State Medical Association 102nd Annual Meeting

THE ALUMNI FOUNDATION

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The Council and the Board of Directors of The University of South Dakota School of Medicine Alumni Foundation join with the 1,711 Alumni and Associate Members of the Foundation in extending our sincere congratulations to the Class of 1983 who received their M.D. Degree on Saturday, May 14, 1983.

Marc N. Aldrich, M.D., Deadwood, SD Raymond H. Allen, M.D., Brookings, SD Neil F. Benson, M.D., Rapid City, SD Gregg W. Carlson, M.D., Mitchell, SD Mark S. Clippinger, M.D., Vermillion, SD Gregory A. Cooper, M.D., Wolsey, SD Kathryn K. Berg Dittes, M.D., Plymouth, MN

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S Medicine

A Simple Method of Treatment of Fractures of the Fifth Metacarpal Neck and Distal Shaft (Boxer's Fracture)*

Robert Van Demark, M.D.*

ABSTRACT

Fractures of the metacarpal neck occur commonly and most frequently at the little finger, as a result of a blow with the clenched fist. The metacarpal head falls forward, with posterior angulation at the fracture site. The stronger flexor structures maintain this position. Numerous complications have been reported with treatment, including stiff fingers, deformity, sores

Fractures of the fifth metacarpal neck are common and account for the majority of all metacarpal neck fractures. Clinically they occur following a blow

Figure 1
Fracture of the distal fifth metacarpal, occurred in a fight.
Note the forward displacement of the head and the posterior prominence.

* Paper presented at Orthopedic Update, September, 1982 at Lake Okoboji, Iowa.

† Orthopedic surgeon, 1301 S. Ninth Avenue, Sioux Falls, SD.

over the knuckles and malunion. Because of the high recurrence of deformity, acceptance of 70 degrees angulation at the fracture site has been recommended.

With a cooperative patient and no associated injury present, a good result without deformity can be achieved by closed methods herein outlined.

with a clenched fist; there is swelling over the dorsum of the distal fifth metacarpal, and the metacarpal head is displaced volarly (Figs. 1, 2). They are basically unstable because of comminution of the anterior cortex. For this reason, and the stronger pull of the flexor structures, there is a tendency for the reduced fracture to settle back in its original angu-



Figure Roentgenogram of fracture.

lated position. Because of the instability and difficulties in maintaining reduction, many techniques have been proposed, including intramedullary nailing with a K-wire through the articular surface of the metacarpal head, open reduction, plate and screw fixation,³ and casting with the fingers bent which can result in pressure sores over the knuckles. All of these methods may result in permanent disability and loss of motion.¹ To avoid them it has been recommended that up to 70 degrees of angulation be accepted at the fracture site if the rotational deformity is minimal.⁵

The little finger points to the scaphoid prominence at the base of the thumb. This fact must be kept in mind in any treatment of the fracture. Failure to do so will result in gross rotational deformity.

In most of these patients local anesthesia is preferred, since the fracture has been an unexpected event. If an ulnar nerve block is done at the wrist, a medial and dorsal subcutaneous nerve block must be done in addition, to block the dorsal cutaneous branches to the little finger and medial half of the ring finger; these nerve branches are given off 2-3 inches above the wrist joint.

We prefer to block the ulnar nerve at the elbow, where it is easily accessible and palpable behind the medical epicondyle. Using a syringe of Marcaine 0.25% (Bupivacaine) and a #25 needle, and with the arm in wide abduction, elbow flexed and externally rotated on an arm board, the injection is made directly from above downward into the groove between the medial epicondyle and the olecranon, the nerve held immobile between two fingers. Paresthesias are easily produced whereupon 5-10 cc. of anesthestic is injected. It is desirable to inject the anesthetic solution around the nerve, rather than directly into the substance of the nerve. Anesthesia in the ulnar nerve distribution requires 15-20 minutes and is easily tested for in the painless ring finger where the ulnar half of the skin is anesthetized, and the radial half has normal sensation.

Reduction of the anesthetized fracture rarely presents a problem. It is easily achieved by a palmar directed pressure over the metacarpal shaft and a dorsally directed dorsal pressure on the angulated head (Figure 3). Rotational alignment is then checked, making sure the tip of the little finger points to the scaphoid tubercle.

With the finger properly aligned and the fracture corrected, alternate strips of ½ inch adhesive tape are placed across the middle of the phalanges to avoid the knuckles, going first around the hand to hold the fracture in position (Fig. 4), and then longitudinally in the U manner to hold the correct alignment (Fig. 5). This basket type of taping is then repeated twice, and a loose circumferential strip of

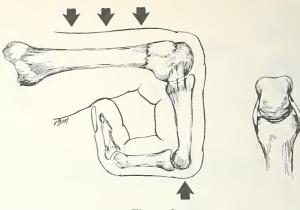


Figure 3 (from Jahss²). Reduction of fracture by palmar directed pressure over the metacarpal shaft and dorsally directed pressure on the angulated head through the phalanges.



Figure 4
Half-inch adhesive tape placed across the middle of the middle phalanx going around the hand to hold fracture in position, with the tip of the finger pointing to scaphoid tubercle. There is no tape or pressure over the knuckles.



Figure 5
Longitudinal taping in U manner to maintain alignment, finger pointing to the tubercle.



Figure 6
Final taping of basket type, secured by loose circumferential taping at the wrist. The knuckles and finger tips are visible.



Figure 7
Final X-ray following reduction and tape immobilization.

tape is placed at the wrist to insure the adhesive strips do not become loose (Fig. 6). Check up X-rays will usually show an excellent position (Fig. 7); should they not, the procedure can be painlessly repeated, since the anesthetic lasts 3-4 hours.

The circulation of the free distal phalanx is always checked and the patient instructed to keep the hand elevated for forty-eight hours. Care must be taken to insure the taping remains intact. The duration of immobilization is 3 weeks. The taping is changed weekly, the skin cleansed with Tape-off and careful-

ly reapplied so as to maintain the correct position as shown on X-rays.

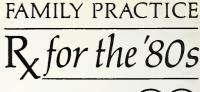
On removal of the taping at the end of 3 weeks, the patient is instructed in gentle active exercises to be performed at hourly intervals. He is encouraged to help with the dishwashing at home three times a day, if whirlpool is not available. These fingers loosen up nicely and the results have been uniformly good in our hands, if the patient is cooperative and there are no associated injuries.

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S Chapter News







SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

FUTURE DIRECTIONS FOR MEDICAL EDUCATION

A Report of the Council on Medical Education Adopted June 15, 1982, by the House of Delegates of the American Medical Association

Part I

Recommendation 1:

The medical profession and those responsible for medical education should strengthen the general or broad components of both undergraduate and graduate medical education. All medical students and resident physicians should have general knowledge of the whole field of medicine regardless of their projected choice of specialty.

Recommendation 2:

Schools of medicine should accept the principle and should state in their requirements for admission that a broad cultural education in the arts, humanities, and social sciences, as well as in the biological and physical sciences, is desirable.

Recommendation 3:

Medical schools should make their goals and objectives known to prospective students and premedical counselors in order that applicants may apply to medical schools whose programs are most in accord with their career goals.

Recommendation 4:

Medical schools should state explicitly in publications their admission requirements and the methods they employ in the selection of students.

Recommendation 5:

Medical schools should require their admissions committees to make every effort to determine that the students admitted possess integrity as well as the ability to acquire the knowledge and skills required of a physician.

Recommendation 6:

Although the results of standardized admission testing may be an important predictor of the ability of students to complete courses in the preclinical sciences successfully, medical schools should utilize such tests as only one of several criteria for the selection of students.

Continuing review of admission tests is encouraged because the subject content of such examinations has an influence on premedical education and counseling.

Recommendation 7:

Medical schools should improve their liaison with college counselors so that potential medical students can be given early and effective advice. The resources of regional and national organizations can be useful in developing this communication.

Recommendation 8:

Medical schools are chartered for the unique purpose of educating students to become physicians and should not assume obligations that would significantly compromise this purpose.

Recommendation 9:

Medical schools should inform the public that, although they have a unique capability to identify the changing medical needs

of society and to propose responses to them, they are only one of the elements of society that may be involved in responding.

Medical schools should continue to identify social problems related to health and should continue to recommend solutions, but they should participate in such solutions only when adequate resources are available, and when there is no hazard of compromising a school's primary purpose.

Recommendation 10:

Medical school faculties should continue to exercise prudent judgment in adjusting educational programs in response to social change and societal needs.

Recommendation 11:

Faculties should continue to evaluate curricula periodically as a means of insuring that graduates will have the capability to recognize the diverse nature of disease, and the potential to provide preventive and comprehensive medical care. Medical schools, within the framework of their respective institutional goals and regardless of the organizational structure of the faculty, should provide a broad general education in both basic sciences and the art and science of clinical medicine.

Recommendation 12:

The curriculum of a medical school should be designed to provide students with experience in clinical medicine ranging from primary to tertiary care in a variety of inpatient and outpatient settings, such as university hospitals, community hospitals, and other health care facilities.

Medical schools should establish standards and apply them to all components of the clinical educational program regardless of where they are conducted. Regular evaluation of the quality of each experience and its contribution to the total program should be conducted.

Recommendation 13:

Medical schools and teaching hospitals should continue to seek diversified financial support for medical education, so that excessive reliance on any single source of income does not jeopardize balanced allocation of resources to teaching, research, and patient care.

Recommendation 14:

Faculties of medical schools have the responsibility to evaluate the cognitive abilities of their students. Extramural examinations may be used for this purpose, but never as the sole criterion for promotion or graduation of a student.

Recommendation 15:

As part of the responsibility for granting the M.D. degree, faculties of medical schools have the obligation to evaluate as thoroughly as possible the non-cognitive abilities of their medical students.

Recommendation 16:

Medical schools should continue to recognize that the instruction provided by volunteer and part-time members of the faculty and the use of facilities in which they practice make important contributions to the education of medical students. Development of means by which the volunteer and part-time faculty can express their professional viewpoints regarding the educational environment and curriculum should be encouraged.

S Medicine

Myxoid Tumor of the Uterus and Right Atrial Myxomas

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ABSTRACT

An unusual case of a young woman with simultaneous myxoid lesions involving the right atrium and uterus is presented. There was considerable clinical difficulty in resolving the relationship of these lesions and in deciding on appropriate management. The additional past history of bilateral

familial adrenal hyperplasia and Cushing's syndrome, an incidental pheocyromocytoma of one adrenal and a peculiar tumor of the retroperitoneum possibly representing a retroperitoneal mesothelioma gave rise to a bizarre constellation of findings worthy of reporting.

INTRODUCTION

This young woman presented with a polypoid myxoid lesion of uterus protruding from the cervical os. Physical examination led to the discovery of multiple cardiac myxomas of the right atrium. The relationship of these lesions represented a clinical puzzle especially in a young woman with a past history of familial bilateral adrenal hyperplasia producing Cushing's syndrome, the finding of an incidental pheochromocytoma of one adrenal and a bizarre retroperitoneal tumor possibly representing a mesothelioma. The morphology of these lesions and clinical course of the patient are described.

REPORT OF A CASE

This 26 year old caucasian female, gravida 4, para

4, married for nine years, presented with the chief complaint of vaginal discharge. All four children were delivered by Cesarean section. One child died of lung problems after birth. The patient had had a tubal ligation after her last Cesarean section. Eight weeks prior to seeing her physician the patient noted a foul-smelling vaginal discharge with occasional blood spotting. Menses prior to that time had been regular each month with a flow of eight days. There was no previous intermenstrual spotting, dysmenor-rhea, or dyspareunia. On pelvic examination, there was a fungating lesion presenting at the cervical os. She was admitted to the hospital for dilatation and curettage.

Past history included removal of both adrenals, one at age 12 and one at age 13, for Cushing's disease. She had been on maintenance steroid therapy since that time. An incidental adrenal pheochromyocytoma was found in one adrenal. The patient also had had a retroperitoneal mesothelioma

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removed. She had a sister two years older who had also had a bilateral adrenalectomy for Cushing's disease one year before her own. There was no history of familial endocrine disease except for an older sister 6'7" in height and another brother 6'11" in height. Neither had acromegalic features. Review of systems was otherwise negative.

Physical Examination

Temperature 99°F, pulse 80/minute and regular, respirations 16/minute and regular, blood pressure 120 systolic and 72 diastolic, weight 53 kilograms, height 168 cm. Examination of the head, neck, breasts, lungs, abdomen, extremities and the nervous system were within normal limits, except for abdominal scars of previous surgery. There was a grade III (six grades) systolic heart murmur over the precordium and upon further questioning the patient admitted to recent episodes of light-headedness, one of which occurred when she was lying in bed with her right side down. A sector ultrasound scan revealed a large, right intra-atrial mass felt to be consistent with a myxoma.

Because of the size of this lesion, as well as the patient's symptoms, immediate surgical resection was recommended. At surgery that same day, when the right atrium was opened, a large 4 cm. friable, pedunculated right atrial tumor, appearing grossly as a myxoma, which had prolapsed through the tricuspid valve into the right ventricle, was found. This was reduced and found to have a stalk about 1 cm. in diameter immediately above the posterior aspect of the inferior vena cava taking origin from the interatrial septum. This was removed with complete excision of the interatrial septum at the base of the tumor with primary closure. There were three other separate and discrete areas of myxomatous tumor. One was 1 cm. in greatest dimension and placed between the coronary sinus and septal leaflet of the tricuspid valve. The second was 0.5 cm. in greatest dimension and located superior and lateral to the coronary sinus. The third was 0.6 cm. in greatest dimension and planted between the coronary sinus and septal leaflet of the tricuspid valve. Each of these was removed by excision of the atrial site of origin down to the epicardium with primary closure. The patient was able to be weaned off cardiopulmonary bypass in a normal sinus rhythm with no difficulty whatsoever. Postoperatively, she required no pressor support initially and the duration of the hospitalization was totally without incident.

One week after the open heart procedure, a dilatation and curettage of the uterus was performed. Myxomatous tissue with blood clot and debris was removed.

One month later the patient was readmitted for

definitive surgery for her uterine lesion. At operation, abdominal and pelvic exploration revealed no abnormality except for a 2 x 2 cm. nodule in the posterior wall of the lower uterine segment, indistinguishable clinically from a small intramural leiomyoma, and a slightly enlarged left obturator lymph node. The uterus was otherwise unremarkable in size or appearance. Frozen section examination of the lymph node revealed chronic lymphadenitis and of the intramural lesion indicated no histopathologic signs of malignancy. The surgery was confined to total abdominal hysterectomy and biopsy of the above mentioned lymph node.

After the removal of the uterus, there was generalized oozing from all the surgical margins and difficulty maintaining hemostasis was encountered. A battery of tests for hemostasis was obtained and all the parameters including prothrombin time, test for fibrin split products, platelet count, partial thromboplastin time and fibrinogen level were abnormal indicating disseminated intravascular coagulation. She was treated with supportive measures, fresh frozen plasma, and cryoprecipitates and approximately four hours later all the hemostatic parameters reverted to normal levels.

Pathology

Grossly the major cardiac lesion was submitted in fragments, measured 5.5×5.2 cm. and was tanyellow, slippery with focal calcification as evidenced by gritty white 0.2 cm. areas on cut section. Separately submitted were three similar lesions measuring $1.5 \times 1.0 \times 1.0$ and 0.8 and 0.3 cm. in greatest dimension.

On the microscopic examination the surface of the largest tumor was irregularly polypoid and lined by cells with prominent nuclei with an occasional conspicuous nucleolus. No mitoses were seen in the tumor. Red cells and fibrin were trapped within the stroma of the main tumor accompanied by occasional clusters of hemosiderin laden macrophages. The lesions were composed of a sparsely cellular stroma with wisps of basophilic material in which embedded round to oval nuclei, which were hyperchromatic but varied little in size or shape. The nuclei were surrounded by eosinophilic cytoplasm, but no cell borders were seen. Large cells with one to four hyperchromatic oval nuclei and wisps of amphophilic cytoplasm giving a stellate appearance were also present. There were numerous vascular spaces lined by prominent endothelial cells. Sections of the separate tumors described above were similar to the main tumor. Colloidal iron stain was diffusely positive in the cytoplasm of the largest lesion. Immunoperoxidase stains for factor VIII were equivocal on all of the lesions (Fig. 1).



Figure 1 View of cardiac myxoma.

Fragments of tissue removed as endometrial curettings revealed necrotic tissue with myxoid features infiltrated with neutrophils and histiocytes. Surgical pathologic consultations with the Armed Forces Institute of Pathology, the division of surgical pathology of the Mayo Clinic and division of surgical pathology of Massachusetts General Hospital, all indicated the cardiac and uterine lesions were not similar. The consultants all felt the cardiac lesions were typical cardiac myxomas, but the nature of the uterine lesion was uncertain.

On pathologic examination, the uterus weighed 117 gms and was 9 x 6 x 4 cm. The external surface was smooth and glistening. The tubes and ovaries



Figure 2
Gross picture of circumscribed myxoid leiomyoma of uterus. The defect in the tumor abuts on the endometrial cavity and was probably created by prior curettage.

were not attached. In the lower uterine segment was an intramural 2.2 x 2 x 1 cm. yellow, well demarcated, gelatinous mass 0.8 cm from the external surface and 0.1 cm. from the internal endometrial cavity (Fig. 2). A V-shaped defect in the tumor was present at the junction with the endometrial surface.



Figure 3
Junction of myometrium and myxoid leiomyoma. Note muscle bundles encircling nonencapsulated tumor.

This was the probable result of the prior curettage. Microscopically the tumor had no capsule and irregular fingers of myxoid tissue extended into the surrounding myometrium (Fig. 3). The myometrium immediately surrounding the tumor did show a different orientation of the muscle fibers than the surrounding myometrium and gave the suggestion that it was part of a leiomyoma. The tumor showed variable cellularity with plump-to-spindle shaped cells with amphophilic pale cytoplasm tailing off into a gray stroma (Fig. 4). The nuclei were uniform,



Figure 4 High power of myxoid tumor of uterus with scattered inflammatory cells H & E $430\times$.

vesicular, oval or round with conspicuous nucleoli. There was minimal pleomorphism or hyperchromatism in the nuclei of the tumor. On several counts 0-1 mitoses per 10/HPF were counted. Focally, there was infiltration with histiocytes, lymphocytes, and segmented neutrophils. Ultrastructural examination of the uterine mass performed by Gail L. Woods showed that the tumor was composed of cells having intracytoplasmic myofilaments and pinocytotic vesicles consistent with a leiomyoma (Fig. 5).



Figure 5
Electron micrograph showing typical smooth muscle cells in uterine tumor.

A repeat consultation with divisions of surgical pathology of the Massachusetts General Hospital and Mayo Clinic indicated that the uterine lesion was a myxoma and not related to the cardiac tumor. One consultant gave an independent opinion without knowledge of the ultrastructural findings that the lesion probably was a myxoid variant of leiomyoma because of the orientation of muscle bundles at the periphery of the myxoid portions of the tumor. Pelvic cell washings showed no evidence of malignancy. The endometrium was proliferative with focal chronic endometritis. The cervix showed chronic cervicitis.

Slides of the previous resected adrenals with hyperplasia and the incidental pheochromocytoma were available but slides of the retroperitoneal lesion were not available.

Discussion

This case proved to be both a clinical and pathologic riddle. This woman presented with vaginal discharge secondary to a necrotic polypoid uterine lesion. Physical examination revealed a heart murmur which subsequently proved to be due to a right

intra-atrial myxoma which was removed surgically. The possibility that the uterine and cardiac lesions were related was entertained since fragments of material removed from the uterus did have myxoid features. Consultation with the Armed Forces Institute of Pathology and surgical pathology departments of Mayo Clinic and Massachusetts General Hospital indicated that this was not the case. Another confusing feature was the fact that there were multiple cardiac myxomatous lesions located in the right atrium rather than the usual single myxoma location in the left atrium.

The circumscribed appearance and lack of atypicality and mitotic activity in the uterine lesion certainly would indicate a benign tumor. There was a suggestion from light microscopic examination that the lesion was a myxoid degeneration of a leiomyoma as smooth muscle bundles at the periphery of the lesion were oriented in a circular fashion around the tumor rather than interdigitating with the remainder of the myometrium. The supposition was confirmed by electron microscopy which indicated the smooth muscle origin.⁵ The possibility that the uterine lesion was a metastasis from the cardiac lesions or, even more unlikely the reverse situation, was initially entertained but rejected after both lesions were fully studied. Although no electron microscopic study was done of the cardiac lesions, they were felt to be morphologically typical of cardiac myxomas and different than the uterine lesion after the latter was fully studied.

Myxoid leiomyosarcomas in elderly women have been infrequently observed, but the present lesion did not have significant mitotic activity and was circumscribed grossly. This, in addition to the absence of metastatic lesions, made us classify the uterine lesion as a benign leiomyoma.

Leiomyomas of the uterus have been estimated to be present in 40% of women over the 35³ years of age or in 20-30% of women older than 30.2 Submucous leiomyomas can certainly present at the cervical os or as necrotic polypoid projections. However, this lady was younger than the usual age group in which this lesion occurs. The tumor of the uterus was also single as opposed to the multiple leiomyomas often seen in older women. Different microscopic appearances including cellular, bizarre (with large irregular giant cells), epithelioid (leiomyoblastoma), and vascular variants of leiomyomas as well as leiomyosarcoma have been well described. Intravenous leiomyomatosis, benign metastatizing leiomyoma and leiomyomatosis peritonealis disseminata have been well described, but do not fit the present lesion. Plexiform tumorlets² or neoplasm simulating gonadal stromal tumors² have also been seen within the uterus, but do not resemble the myxoid tumor in this case.

Myxoid degeneration has been described² in uterine leiomyomas, but is less discussed than the more usual changes in leiomyomas such as calcification, hemorrhage, necrosis, cyst formation, fatty change and hyalinization. Norris and Zaloudek² mention and illustrate myxoid degeneration of leiomyoma, but the changes are not nearly as marked as in this present case. Several authors,^{7, 8} have described marked edema seen in leiomyomas in pregnancy and after the administration of a potent progestin. However, most of the changes described progress to hemorrhage, necrosis, hyalinization and fibrosis and appear to be different than that seen in this case.

The etiology of the transient intraoperative disseminated intravascular coagulation is unclear. The fact that it happened during the hysterectomy and not during the open heart operation makes one suspect a transient thromboplastin infusion from the uterus or the myxoid tumor may have precipitated the hemor-

rhagic event.

The presence of a myxoid change in a leiomyoma presenting in a young woman who also had a history of right atrial myxomas and history of bilateral adrenal hyperplasia, pheochromocytoma and a retroperitoneal mesothelioma represents a combination of unusual phenomena. This case also illustrates how electron microscopy can be used to elucidate a lesion not fully understood by light microscopy.

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BRIEF SUMMARY PROCARDIA " (nifedipine) CAPSULES

INDICATIONS AND USAGE: f. Vasospastic Angina: PROCAROIA (nitedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria 1) classical pattern of angina at rest accompanied by ST segment elevation. 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant lixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm

in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents. In chromic stable angina (effort-associated angina) PROCAROIA has been effective in controlled trails of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available inforbeta blocking agents may be beneficial in patients with chronic stable angina, but available infor-mation is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When in-troducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.) CONTRAINDICATIONS: Known hypersensitivity reaction to PRDCARDIA. WARNINGS: Excessive Hypotension. Although in most patients, the hypotensive effect of PRDCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tol-erated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Subsequent byward occurs in patients severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PRDCARDIA and a beta blocker, but the possibility that it may occur with a combination of PRDCARDIA and a beta blocker. But the possibility that it may occur with due to the combination of PRDCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of tentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased trequency duration or seventy of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased disable pressure with prepased heart calle, or from prepased demand.

associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone. Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catechol-amines Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCAROIA. Patients with tight aortic stenosis may be at greater risk for

such an event

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and litration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vaso-dilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Orug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

literature reports suggesting that the combination may increase the likelihood of congestive heart tailure, severe hypotension or exacerbation of angina. Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination. Oigitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility. When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

man dose
Pregnancy Category C. Please see full prescribing information with reference to teratogenicity in

Pregnancy Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

AOVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients. transient hypotension in about 15%, paliphtation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported muscle cramps, nervousness, dyspinea, nasal and chest congestion, diarrhea, constitution, inflammation, joint stiffness, shakness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension. In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these natures. It remains possible, however, that some or many of

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventroular arrhythmas or conduction disturbances each occurred in thewir than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase. CPK, LOH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease atter about eleven months of intedipine therapy. The relationship to PROCAROIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCAROIA therapy, has been reported twice in the extensive world literature.

Interature

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine

PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069
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3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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S Medicine

Pulmonary Embolus with a Normal Ventilation Perfusion Lung Scan: Case Report

Leonard M. Gutnik, M.D.*

The patient is a 44 year old white female who was admitted to the hospital with the right nucleus pulposus syndrome at C5-6. The patient had an anterior cervical fusion. I was asked to see her when she developed severe chest pain. She described nausea, heat, and burning central chest pain with sharp pains under the left breast. These pains were worse when

she took a deep breath. There were no leg symptoms of significance. Her past history of significance included pulmonary embolus after gallbladder surgery two years previously and a two packs per day cigarette habit. The physical examination and Doppler examination of the extremities were within normal limits, as were the chest x-ray and EKG. The

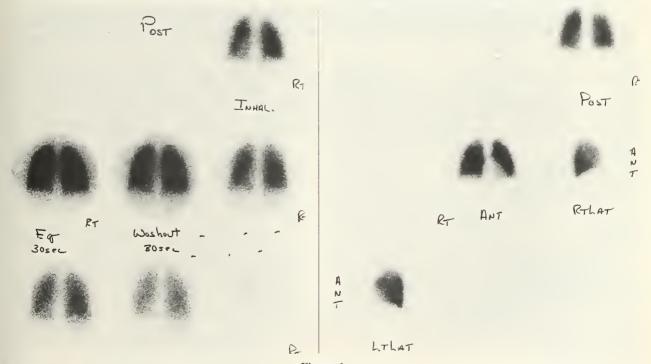


Figure 1
Normal radionuclide ventilation scan on left and perfusion scan on right.

ventilation perfusion lung scan was normal (Figure 1). The blood gases showed an increased A-a gradient (pO₂-62, pCO₂-29).

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Because the history was strongly suggestive of pulmonary emboli, despite the negative non-invasive studies, a pulmonary angiogram was ordered and completed within several hours of the radionuclide scan. The patient was noted to have an allergy to kidney dye manifested by hives (Figure 2). She was premedicated with steroids, and a pulmonary arteriogram was done. ^{1, 2} This study showed a large



Figure 2
Cutoff of right main pulmonary artery due to obstruction on angiogram.

thrombus in the right mainstem pulmonary artery extending into the origin of the right upper lobe. The embolus measured 1cm in length and was obstructing approximately 80% of the right mainstem pulmonary artery orifice. The patient was transferred to the intensive care unit and because of the degree of obstruction and pain, she was started on streptokinase drip. Patient did well and was switched to heparin; one week later, she developed right flank pain, and a CT scan of the abdomen was compatible with retroperitoneal bleeding. Under these circumstances, a vena caval umbrella was placed. The patient slowly improved and heparin was restarted. The patient was converted to coumadin and discharged from the hospital. The follow-up has been over one year, and to date her course has been without difficulties.

DISCUSSION

This particular case is interesting from several standpoints:

1) A pulmonary embolus with a normal ventilation perfusion lung scan.

- 2) The use of streptokinase and heparin for the treatment of pulmonary embolus.
- 3) The placement of a vena caval umbrella and its implications.

I will primarily speak to the first issue, but will address briefly the other items.

Pulmonary embolus with a normal ventilation perfusion lung scan is a rarity.^{3, 4, 5} A pulmonary embolus is usually the result of a deep venous thrombosis that has dislodged from the venous wall and traveled upstream in the venous channels to the lungs. The physical signs and symptoms are myriad and frequently unreliable. I wish to focus my attention to the ventilation perfusion scan. There are many techniques for this procedure. Commonly perfusion scanning is done with venous infusion of a radionuclide labelled microsphere of 20-40 microns in diameter. These spherish emboli are distributed uniformly in the lungs where the pulmonary arterioles act as a sieve to the particulate microspheres. The amount of microsphere accumulation will be directly proportional to the pulmonary artery blood flow. Usually six views are obtained: anterior, posterior, both laterals and both posterior obliques. The ventilation scan is done usually with Xenon¹³³. It is inhaled and measured with a gamma camera. Serial pictures are taken frequently to measure washout. There exists significant controversy in which portion of this test to be performed first. Normal perfusion study usually rules out a pulmonary embolus. At least two segmental defects in perfusion with a normal ventilation scan is considered a high probability scan for pulmonary embolus. Defects in perfusion with matching ventilation defects are considered low probability of pulmonary embolus, since any pulmonary process, causing decreased ventilation such as pneumonia, asthma or obstructive lung disease, will also produce decreased pulmonary perfusion secondary to pulmonary vasoconstriction from diminished oxygen. Intermediary scan regions also exist.8

I believe that no testing in medicine is absolute. Probability is just that and subject to error. While reviewing this particular arteriogram, the embolus was 80% obstructing the origin of the right mainstem pulmonary artery. One could see the dye flowing around the obstruction, and I suspect this accounts for the normal ventilation perfusion scan. There have been several articles discussing the accuracy and sensitivity of ventilation perfusion lung scanning. ^{7, 9, 10} It is my contention that in this age of medical sophistication, we must rely on our judgment to integrate the various test results into our patient care model.

The use of streptokinase and heparin for the treat-

ment of pulmonary embolus and/or deep venous thrombosis is well documented, and I will not speak of these issues here. 11, 12, 13 However, I wish to point out that with this patient's prior surgery, both agents were relatively contraindicated.

The placement of a vena caval umbrella carries approximately 15% incidence of severe lower extremity edema^{14, 15} and is only a temporary measure. ^{14, 15} We were fortunate that this particular patient did well and has to date shown none of its potential side effects.

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S Department of Health

Nitrous Oxide, A Health Hazard??

In a current surgery textbook utilized by medical students and practicing physicians it is stated, "Although the perfect anesthetic agent does not exist at the present time, nitrous oxide (N₂O) probably comes closer than any other agent to being an ideal anesthetic. It is completely nontoxic. It has no clinically significant effect upon respiration, on the cardiovascular system, on metabolism, on renal function, or on hepatic function."

During the past few years articles, primarily in the anesthesia literature, have cited the hazards of waste anesthetic gases in anesthetists and persons exposed to them in surgery, in laboratories, and in dental offices. ²⁻⁸ Most reports pertained to the findings in specific groups, i.e., anesthetists, who were exposed to multiple different anesthetic agents, precluding identification of the effect of specific agents.

A recent communique from George G. Dudley, D.D.S., Chief, Dental Health Section, Division of Health Services, Raleigh, N.C., to Carlos Lozano, D.D.S., President, Association of State and Territorial Dental Directors,9 focused attention upon the hazard of occupational exposure to N2O, citing an epidemiological study of 30,650 dentists and 30,547 chairside assistants for the effect of occupational exposure to N₂O.⁹ It showed that in dental chairside assistants there were twice as many spontaneous abortions, and they produced one and one half times as many children with congenital anomalies as would be expected. Also, the wives of male dentists surveyed had a statistically significant increase in the number of spontaneous abortions. References cited from the literature indicated that teratogenic, abortifacient, and the spermatogenic effects of N₂O are corroborated by animal experimentation. 10, 11

The maximum safe level of N₂O in the work place recommended by the National Institute of Occupational Safety and Health (NIOSH) is 25 parts per million (ppm). ¹² Based upon data from a survey of North Carolina dental offices for N₂O concentrations in the ambient air, it was apparent that excessive levels of anesthetic gases existed in all offices surveyed, regardless of the presence of scavaging apparatus. Waste gas levels of N₂O ranged from 200 to 2,318 parts per million (ppm). ⁹ In addition to the excessive N₂O levels in the dental offices, concentrations in adjacent hallways and adjoining rooms far exceeded the minimum recommended levels.

By using efficient waste gas scavagers, lower gas flow, and closed circuit anesthesia units rather than open or semi-open systems usually employed in dental offices, exposure to waste anesthetic gases is much less in the surgery theatre than in dental offices. However, due to leaks in the gas delivery system, measurable levels of waste anesthesia gases can easily be present in a surgery theatre and are a potential hazard in any operating room.

Because N_2O , previously thought to be innocuous, has been implicated as teratogenic, abortifacient, and spermotoxic, it must be added to the list of substances to be controlled in the workplace. Unfortunately, there are no legally enforcable state or federal standards to regulate the occupational exposure to waste N_2O gas.

Measures to prevent adverse effects of N₂O upon those working in environments potentially contaminated by waste gases include, 1) monitoring of ambient waste gas levels, maintaining a level not to exceed 25 ppm N₂O, 2) insofar as possible, avoidance of exposure to N₂O during the first trimester of pregnancy, 3) monitoring and data collection relating to adverse effects upon reproduction of personnel exposed to waste gases, as an indicator of the success of efforts to regulate waste gas exposure, 4) education of all individuals potentially exposed to N₂O over an extended interval regarding the effects of this agent upon the reproductive mechanism, with recommendation that unnecessary exposure to N₂O be avoided.

John B. Gregg, M.D. Willis Stanage, M.D. Office of Medical Services South Dakota Department of Health

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SD

This Is Your Medical Association

Loren Amundson, M.D., Sioux Falls, has been elected to serve a 5 year term on the Board of Directors of the American Board of Family Practice.

Dr. Amundson, a native of Colton, SD, was founding chairman of the Department of Family Medicine at the USD School of Medicine, occupying that position from 1974 to 1979. Currently he is a Professor of Family Practice at the School of Medicine and Associate Director of the Sioux Falls Family Practice Residency Program. He has served as secretary-treasurer of the South Dakota Academy of Family Physicians for the last 12 years. He is a delegate to the American Academy of Family Physicians and serves on its Commission on Education. He is also a diplomate of the American Board of Family Practice.

* * * *

The American College of Physicians announced that **Jerome W. Freeman, M.D.,** Sioux Falls, has been elected to Fellowship.

* * * *

Dr. Linda Peterson, M.D., Watertown, spoke and conducted a workshop on gynecology at this year's South Dakota state convention of licensed practical nurses held in Watertown. **Dr. David Elson,** Sioux Falls, also held a workshop on medical oncology. In the evening, **Dr. Paul Larson,** Watertown, spoke and showed slides on Madagascar.

* * * *

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Drs. Steven Vosler, Spearfish; Lambert W. Holland, Chamberlain; Jack Berry, Mitchell; S. W. Allen, Jr., Watertown; T. J. Wrage, Jr., Watertown; and Lonnie Waltner, Bridgewater, have completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

* * * *

Charles Swanson, M.D., Pierre, spoke at the consumer health education program sponsored by St. Mary's Hospital, Pierre. Dr. Swanson discussed the effects of smoking on the human body.

* * * *

The 1983 University of South Dakota Alumni Achievement Award was presented to **Duane B. Reaney, M.D.,** Yankton, during commencement exercises. Dr. Reaney received his BS in medicine from USD in 1945. He has practiced medicine in Yankton for 35 years and has been on the faculty of the Medical School since 1963. He is a past president of the South Dakota State Medical Association.

* * * *

L. C. Askwig, M.D., Pierre, has announced his retirement. Dr. Askwig began his practice in Pierre 36 years ago.

continued from page 20

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S

Future Meetings

August

- Comprehensive Industrial Hygiene Review Course, St. Paul, MN, Aug. 8-12. Fee: \$600. Credits. Contact: Ruth K. McIntyre, Dir., Cont. Ed., Midwest Ctr. for Occupational Hlth. & Safety, 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- 1983 Black Hills Summer Seminar, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 11-13. Fee: \$100. 15 hrs. AAFP & AMA Category I credits. Contact: L. H. Amundson, M.D., 3001 S. Holly, Sioux Falls, SD 57105. Phone: (605) 335-5008.
- Workshop in Medical Office Management, Radisson South, Minneapolis, MN, Aug. 22-26. Fee: \$500. Contact: Conomikes Assoc., 4270 Promenade Way, Marina del Ray, CA 90291. Phone: (800) 421-6512.
- Medical Malpractice Seminar, The Homestead, Hot Springs, VA, Aug. 26-28. Fee: \$275. Contact: Jeanette Stone, Southern Med. Assoc., P. O. Box 2446, Birmingham, AL 35201. Phone: (205) 323-4400.

September

First Annual Graduate Occupational Health and Safety Institute, Earle Brown Cont. Ed. Ctr., U. of MN, St. Paul, MN, Sept. 12-23. Fee: \$400. 3 credits per course. Contact Ruth K. McIntyre, Dir., Cont. Ed., Midwest Ctr. for Occupational Hlth. & Safety, 640 Jackson St., St. Paul, MN 55101. Phone (612) 221-3992.



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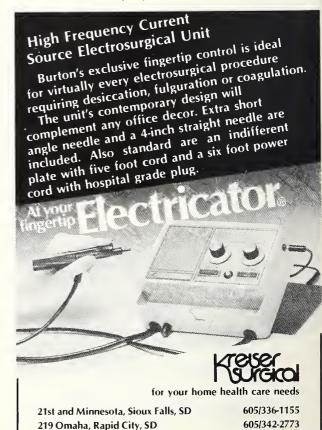
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- Radiology '83, Willey Hall, U. of Minn., Minneapolis, MN, Sept. 12-16. Fee: \$400. 28 hrs. Category I credits. Contact: Lori Wheatcroft, CME, U. of Minn., P.O. Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Pediatric Update for Primary Care Physicians, Saint Paul Hotel, St. Paul, MN, Sept. 16-17. AMA Category I credits. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- 2nd Annual McKennan Toxicology Conference, Town House Convention Ctr., Sioux Falls, SD, Sept. 24. Contact: Dr. Brad Wallenberg, McKennan Pharm., 800 E. 21st St., Sioux Falls, SD 57101. Phone: (605) 339-7873.

October

- Eighth Annual Perinatal Conference, Ramada Inn, Sioux Falls, SD, Oct. 17-18. 13 hrs. credit applied for. Sponsored by SD Perinatal Assoc. and USD School of Med. Contact: Margo Varcoe, RN, SD Perinatal Assoc., 1100 S. Euclid Ave., P.O. Box 5039, Sioux Falls, SD 57117-5039. Phone (605) 333-7193.
- **49th Annual Scientific Assembly**, Hyatt Regency Hotel, Chicago, IL, Oct. 23-27. Contact: Dept. of Education, Am. College of Chest Phys., 911 Busse Highway, Park Ridge, IL 60068. Phone: (312) 698-2200.
- 51st Annual Postgraduate Assembly, Red Lion Inn, Omaha, NE, Oct. 31-Nov. 2. Contact: Lorraine Seibel, Omaha Mid-West Clinical Society. 7363 Pacific St., #210-A, Omaha, NE 68114. Phone: (402) 397-1443.



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Transactions Of The South Dakota State Medical Association **102nd Annual Meeting**

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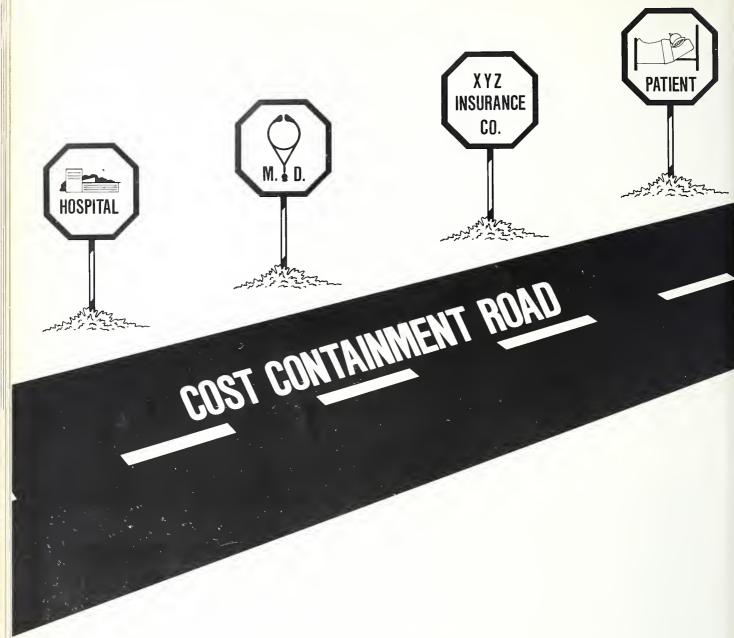
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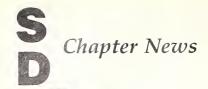
SCIENTIFIC ARTICLES

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NEXT MONTH

The Rural Family Medicine Clerkship (RFMC) at the University of South Dakota School of Medicine — A Six Year Review, 1977-1982

Clinicopathological Conference Seventy-Four Year Old Caucasian Female with Filling Defect in Cecum by X-ray and Blood Loss Anemia



R for the 80s





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

FUTURE DIRECTIONS FOR MEDICAL EDUCATION

A Report of the Council on Medical Education Adopted June 15, 1982, by the House of Delegates of the American Medical Association

Part II

Recommendation 17:

Each medical school should establish, or review already established, criteria for the initial appointment, continuation of appointment, and promotion of all categories of faculty. Regular evaluation of the contribution of all faculty members should be conducted in accordance with institutional policy and practice.

Recommendation 18A:

Faculties of medical schools should reevaluate the current elements of their fourth or final year with the intent of increasing the breadth of clinical experience through a more formal structure and improved faculty counseling. An appropriate number of electives or selected options should be included.

Recommendation 18B:

Counseling of medical students by faculty and others should be directed toward increasing the breadth of clinical experience in the fourth or final year. Students should be encouraged to choose experiences in disciplines that will not be an integral part of their projected graduate medical education.

Recommendation 19:

Directors of residency programs should not permit medical students to make commitments to a residency program prior to the final year of medical school.

Recommendation 20:

The first year of postdoctoral medical education for all graduates should consist of a broad year of general training.

For physicians entering residencies in internal medicine, pediatrics, and general surgery, postdoctoral medical education should include at least four months of training in a specialty or specialties other than the one in which the resident has been appointed. (A residency in family practice provides a broad education in medicine because it includes training in several fields.)

For physicians entering residencies in specialties other than internal medicine, pediatrics, general surgery, and family practice, the first postdoctoral year of medical education should be devoted to one of the four above-named specialties or to a program following the general requirements of a transitional year stipulated in the "General Requirements" section of the "Essentials of Accredited Residencies."

A program for the transitional year should be planned, designed, administered, conducted, and evaluated as an entity by the sponsoring institution rather than by onc or more departments. Responsibility for the executive direction of the program should be assigned to one physician whose responsibility is the administration of the program. Educational programs for a transitional year should be subjected to thorough surveillance by the appropriate accrediting body as a means of assuring that the content, conduct, and internal evaluation of the educational program conform to national standards. The impact of the transitional year should not be deleterious to the educational programs of the specialty disciplines.

Recommendation 21:

The Accreditation Council for Graduate Medical Education, individual specialty boards, and respective residency review committees should improve communication with directors of residency programs because of their shared responsibility for programs in graduate medical education.

Recommendation 22:

Specialty boards should be aware of and concerned with the impact that the requirements for certification and the content of the examination have upon the content and structure of graduate medical education. Requirements for certification should not be so specific that they inhibit program directors from exercising judgment and flexibility in the design and operation of their programs.

Recommendation 23:

An essential goal of a specialty board should be to determine that the standards that it has set for certification continue to assure that successful candidates possess the knowledge, skills, and the commitment to upgrade continually the quality of medical care.

Recommendation 24:

Specialty boards should endeavor to develop a concensus concerning the significance of certification by specialty and publicize it so that the purposes and limitations of certification can be clearly understood by the profession and the public.

Recommendation 25:

The importance of certification by specialty boards requires that communication be improved between the specialty boards and the medical profession as a whole, particularly between the boards and their sponsoring, nominating, or constituent organizations and also between the boards and their diplomates.

Recommendation 26

Specialty boards should consider having members of the public participate in appropriate board activities.

Recommendation 27:

Specialty boards should consider having physicians and other professionals from related disciplines participate in board activities

Recommendation 28:

The American Medical Association recommends to state licensing authorities that they require individual applicants, to be eligible to be licensed to practice medicine, to possess the degree of Doctor of Medicine or its equivalent from a school or program that meets the standards of the Liaison Committee on Medical Education or accredited by the American Osteopathic Association, or to demonstrate as individuals, comparable academic and personal achievements.

All applicants for full and unrestricted licensure should provide evidence of the satisfactory completion of at least one year of an accredited program of graduate medical education in the United States. Satisfactory completion should be based upon an assessment of the applicant's knowledge, problem-solving ability, and the clinical skills in the general field of medicine.

The American Medical Association recommends to legislatures and governmental regulatory authorities that they not impose requirements for licensure that are so specific that they restrict the responsibility of medical educators to determine the content of undergraduate and graduate medical education.

Transactions Of The South Dakota State Medical Association 102nd Annual Meeting June 2, 3, 4, 5, 1983

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Jay Hubner, M.D., Chairman (1985) Yankton Lawrence Finney, M.D. (1984) Sioux Falls Richard Hockett, M.D. (1984) Mitchell Wm. G. H. Huet, M.D. (1984) Huron Karl Kosse, M.D. (1984) Aberdeen William Quick, M.D. (1984) Yankton Louis Hogrefe, M.D. (1985) Gregory Clark Likness, M.D. (1985) Watertown J. F. Barlow, M.D. (1985) Sioux Falls Allan Hartzell, M.D. (1985) Sioux Falls Richard Holm, M.D. (1986) Brookings William Dendinger, M.D. (1986) Vermillion Lawrence Massa, D.O. (1986) Sturgis Werner Klar, M.D. (1986) Fort Meade R. E. Van Demark, M.D. (1986) Sioux Falls Student Member

COMMISSION ON MEDICAL SERVICE

Loyd Wagner, M.D., Chairman (1984) Sioux Falls Michael Ferrell, M.D. (1984) Sioux Falls John Knecht, M.D. (1984) Martin P. K. Aspaas, Jr., M.D. (1984) Sioux Falls Tom Huber, M.D. (1984) Pierre E. T. Ruud, M.D. (1985) Hot Springs Kennon Broadhurst, M.D. (1985) Aberdeen Curtis Wait, M.D. (1985) Brookings Anthony Petres, M.D. (1985) Salem John Hoskins, M.D. (1985) Sioux Falls Lowell Swisher, M.D. (1986) Kadoka J. A. Rud, M.D. (1986) Rapid City Lee Ahrlin, M.D. (1986) Rapid City Tony Berg, M.D. (1986) Winner Jay Bachmayer, M.D. (1986) Aberdeen Student Member

COMMISSION ON SCIENTIFIC MEDICINE

A. J. Janusz, M.D., Chairman (1985) Aberdeen Loren Amundson, M.D. (1984) Sioux Falls G. Robert Bell, M.D. (1984) DeSmet Julie Stevens, M.D. (1984) Yankton Robert Talley, M.D. (1984) Sioux Falls George Jenter, D.O. (1984) Sturgis Harvey Hart, M.D. (1985) Aberdeen Robert Raszkowski, M.D. (1985) Sioux Falls V. V. Volin, M.D. (1985) Sioux Falls Gene Koob, M.D. (1985) Sioux Falls David Elson, M.D. (1986) Sioux Falls Beth Johnson, M.D. (1986) Sioux Falls Lewis Ofstein, M.D. (1986) Sioux Falls Wm. Tschetter, M.D. (1986) Rapid City Gilbert English, M.D. (1986) Sioux Falls Student Member

PROFESSIONAL LIABILITY COMMISSION

Morris Radack, M.D., Chairman (1984) Yankton Dale Berkebile, M.D. (1984) Rapid City E. W. Sanderson, M.D. (1984) Sioux Falls James Hovland, M.D. (1986) Aberdeen Laura Hays, M.D. (1986) Mitchell Donald Kelley, M.D. (1986) Rapid City Frank Alvine, M.D. (1985) Sioux Falls Jerry Walton, M.D. (1985) Sioux Falls Richard Wake, M.D. (1985) Brookings

CREDENTIALS COMMISSION AND EXECUTIVE COMMISSION

Joseph Hamm, M.D., Sturgis Howard Saylor, Jr., M.D., Huron Richard Gere, M.D., Mitchell W. O. Rossing, M.D., Sioux Falls Gerald E. Tracy, M.D., Watertown Bruce Lushbough, M.D., Brookings Robert Ferrell, M.D., Rapid City Arthur J. Barrett, M.D., Rapid City Durward Lang, M.D., Omaha

GRIEVANCE COMMISSION

Duane B. Reaney, M.D., Chairman (1985) Yankton Russell Harris, M.D. (1984) Rapid City Winston B. Odland, M.D. (1986) Aberdeen Bruce Lushbough, M.D. (1987) Brookings Durward Lang, M.D. (1988) Omaha

LONG RANGE PLANNING COMMITTEE

Gerald Loos, M.D., Chairman (1985) Sioux Falls W. Nicol Guddal, M.D. (1986) Watertown David Smith, M.D. (1986) Yankton Richard Honke II, M.D. (1985) Wagner R. G. Nemer, M.D. (1984) Gregory Michael Haley, M.D. (1984) Mitchell

ARCHIVES AND HISTORY COMMISSION

C. J. McDonald, M.D., Chairman (1984) Sioux Falls C. B. McVay, M.D. (1984) Yankton David Buchanan, M.D. (1984) Huron Virginia Tracy (1984) Auxiliary Roscoe Dean, M.D. (1984) Wessington Springs

LIAISON COMMITTEE

Frank Messner, M.D., Chairman (1984) Yankton Robert Ferrell, M.D. (1984) Rapid City George Thompson, D.O. (1984) Gregory James Monfore, M.D. (1984) Miller Winston Odland, M.D. (1984) Aberdeen

S.D. HUMAN SERVICES CENTER MEDICAL ADVISORY COMMITTEE

Thomas Willcockson, M.D. (1984) Yankton Loyd Wagner, M.D. (1984) Sioux Falls Kenneth Halverson, M.D. (1984) Yankton Richard Renka, M.D. (1984) Rapid City

REPORT OF THE BUDGET AND AUDIT COMMITTEE

June 2, 1983

Ramada Inn, Sioux Falls, SD

The meeting was called to order at 8:30 a.m. by Jay Hubner, M.D., chairman of the Budget and Audit Committee. Present for roll call were Drs. Hubner, Durward Lang, Joseph Hamm, Howard Saylor, William Rossing, Richard Gere, G. E. Tracy, Russell Harris and Mr. Robert Johnson and Mrs. Patty Butler.

The committee reviewed the CPA audit prepared by McGladrey Hendrickson & Company. Mr. Johnson discussed various statements included in the audit and answered questions from the committee. Dr. Tracy then moved that the Budget and Audit Committee approve the audit. The motion was seconded by Dr. Lang and carried.

The meeting adjourned at 9:30 a.m.

FIRST COUNCIL MEETING MINUTES

10:00 a.m. Roosevelt Room, Ramada Inn Thursday, June 2, 1983 Sioux Falls, SD

The meeting was called to order by Richard Gere, Chairman. Those present for roll call were Doctors Durward Lang, Joseph Hamm, Howard Saylor, W. O. Rossing, Bruce Lushbough,

Richard Gere, G. E. Tracy, Russell Harris, Jay Hubner, G. Robert Bartron, A. A. Lampert, Jr., David Buchanan, Guy Tam, Larry Sittner, Lowell Hyland, Frank Messner, R. I. Porter, James Jackson, Robert Ferrell, M. George Thompson, James F. Wunder, Michael Pekas. Others in attendance included Doctors J. B. Gregg, R. H. Quinn, Duane Reaney, James Ryan and Winston Odland.

Dr. Saylor moved that the minutes of the previous meeting be approved as printed and distributed. The motion was

seconded and carried.

I. REPORT OF THE LIAISON COMMITTEE Dr. Ferrell presented the report of the Liaison Committee. Dr. Hamm moved to accept the report of the Liaison Committee. The motion was seconded and carried.

II. EXECUTIVE COMMISSION CONFERENCE CALL Mr. Johnson reviewed the resignation situation of some of the chairmen at the USD School of Medicine. The resignations were prompted because of an unwillingness on the part of the President of USD to promote faculty unless excellence had been demonstrated in education, service and research. The chairman felt it was unfair to require research since faculty had been recruited with emphasis on education and service, not research.

Following discussion, Dr. Tracy moved that a resolution reaffirming the purpose and objectives of the USD School of Medicine be drafted by the appropriate reference committee for introduction to the House of Delegates with a copy of this letter to be sent to the USD President, the Board of Regents and the Governor. The motion was seconded and carried.

III. LETTER FROM NORTH DAKOTA BLUE SHIELD Dr. Saylor moved that the President of the SDSMA appoint a physician to represent South Dakota on the Medical Claims Review Committee of Blue Shield of North Dakota. The motion was seconded and carried.

IV. REQUEST FOR CO-SPONSORSHIP OF HOSPITAL ASSOCIATION SEMINARS

Dr. Lang moved that the SDSMA co-sponsor, without financial involvement, the Hospital Association's seminar dealing with the implications of DRG Reimbursement for hospital-physician relations. The mo-

tion was seconded and carried.

V. ELECTION OF SODAPAC BOARD OF DIRECTORS Dr. Saylor moved that the SDSMA elect the following to the SoDaPAC Board of Directors: W. R. Taylor, M.D., Harvey Hart, M.D., Mrs. Marie Hovland, T. J. Wrage, Jr., M.D., Curtis Wait, M.D., Mrs. Barbara Wait, J. B. Davis, M.D., Robert Hohm, M.D., Michael Haley, M.D., Mrs. Boots Mabee, Courtney Anderson, M.D., Les Kinstad, Karl Wegner, M.D., R. I. Porter, M.D., Mrs. Marlys Porter, N. R. Whitney, M.D., A. J. Barrett, M.D., Mrs. Mary Ann Harris, Robert D. Bloemendaal, M.D., Raymond Nemer, M.D., James Wunder, M.D., L. F. Nelson, M.D.; and ask the SoDaPAC Board of Directors to elect appropriate people to fill the following positions:

District # 1 — 1 Auxiliary

District # 1 — 1 Auxiliary
District # 2 — 1 Auxiliary
District # 4 — 1 Auxiliary
District # 5 — 1 Auxiliary
District # 7 — 3 Physicians, 5 Auxiliary
District # 8 — 1 Auxiliary, 1 Physician

District # 8 — 1 Auxiliary, 1 Physician
District # 9 — 2 Auxiliary
District #10 — 1 Auxiliary

District #11 — 1 Auxiliary

District #12 — 1 Auxiliary

The motion was seconded and carried.

VI. MEDICAL GROUP MANAGEMENT ASSOCIATION RESOLUTION

Dr. Land moved to approve the following resolution to American Family Insurance as adopted by the South Dakota Clinic Managers on May 6, 1983:

WHEREAS, American Family Insurance advertises to prospective policyholders a usual and customary health insurance policy and proclaims to pay all covered expenses in full

WHEREAS, American Family utilizes a formula to compute their usual and customary payment, which very often leaves a substantial portion of the physician's usual and customary charge unpaid and,

WHEREAS, American Family Insurance utilizes a letter notifying physicians of payment under their insurance contract which states that the company is prepared to defend the policy-holder against any legitimate collection efforts used by the physician's office and,

WHEREAS, this letter is offensive to reputable physicians and their legitimate right to receive fair and adequate compensation for their services and,

WHEREAS, this type of communication with our patients creates a feeling of mistrust directed at both the insurance company and the

physician.

NOW THEREFORE BE IT RESOLVED that the South Dakota Medical Group Management Association and the South Dakota State Medical Association strongly encourage American Family Insurance to revise their policyholders communication to eliminate any inference that the physician has overcharged the patient and, BE IT FURTHER RESOLVED that American Family

Insurance revise its advertising so that it will more accurately reflect the benefits actually available under the policy in an attempt to eliminate any confusion or false hopes

on the part of our patients.

The motion was seconded and carried. VII. INTRODUCTION OF STEVE ELLWING

Steve Ellwing of the AMA was introduced to the Council.

VIII. AMA UPDATE ON JCAH STANDARD A confirmation of the AMA's position concerning JCAH standards was presented for the information of the Coun-

IX. HONORARY LIFE MEMBERSHIP

Dr. Saylor moved to accept Dr. Leroy C. Askwig as an honorary life member of the South Dakota State Medical Association. The motion was seconded and

X. AMA POSITION REGARDING THERAPEUTIC USE OF MARIJUANA

Dr. Hamm moved to support the AMA's position regarding therapeutic applications for marijuana and the AMA's recommendation that appropriate regulations and guidelines be established to ensure that bona fide research is carried out, and that medical use beyond the context of clinical investigation is not permitted in the therapeutic use of marijuana. The motion was seconded and carried.

XI. MEDICAID RULES

Bob Johnson reviewed the topic of mandatory substitution of brand name drugs with generic drugs. Following discussion, Dr. Lushbough moved that the Executive Commission be empowered to review the final draft of the rules dealing with the mandatory substitution of generic drugs for Medicaid patients to determine what, if any, action is necessary. The motion was seconded and carried.

XII. BOARD OF MEDICAL EXAMINERS

Dr. Bartron discussed the number of applications for nurse practitioners and midwives and the parameters of their practice. Dr. Tracy moved to refer this matter to the appropriate commission for review and consideration of practice parameters for nurse practitioners and nurse midwives. The motion was seconded and car-

Bob Johnson announced that Kevin Loge will be leaving the staff of the Medical Association and will be joining South Dakota Blue Shield.

The meeting adjourned at 11:00 a.m.

SECOND COUNCIL MEETING MINUTES

11:00 a.m. Roo Sunday, June 5, 1983

Roosevelt Room, Ramada Inn Sioux Falls, SD

The meeting was called to order at 11 a.m. by Richard Gere, Chairman. Those present for roll call: Doctors Joseph Hamm, Howard Saylor, Richard Gere, W. O. Rossing, Durward Lang, A. J. Barrett, G. E. Tracy, J. A. Eckrich, Jr., Granville Steele, James Larson, A. A. Lampert, Jr., David Buchanan, Michael Pekas, R. E. Gunnarson, John Ochsner, R. I. Porter, Frank Messner, Roger Millea, Robert Ferrell, James Jackson, M. George Thompson, James F. Wunder, Daniel Kennelly, Eldon Bell.

Dr. Saylor moved that the minutes of the previous meeting not be read inasmuch as they will be published and distributed. The motion was seconded and carried.

I. INTRODUCTION OF NEW COUNCILORS
The following new councilors were introduced and welcomed to the Council: Granville Steele, M.D., Aberdeen;
James Larson, M.D., Watertown; John Ochsner, M.D.,
Sioux Falls; and Richard Tschetter, M.D., Sioux Falls.

II. ELECTION OF COUNCIL CHAIRMAN Dr. Wunder nominated Dr. Robert Ferrell as chairman of the Council. The nomination was seconded. Dr. Tracy moved that nominations cease and that a unanimous vote be cast for Dr. Ferrell. The motion was seconded and carried.

III. REPORT OF THE ARCHIVES COMMITTEE
Dr. Buchanan reviewed the report of the Archives Committee. Dr. Buchanan moved that all the archives material regarding the South Dakota State Medical Association be placed in the Center for Western Studies located on the campus of Augustana College, with the SDSMA maintaining legal ownership and the Center having custodial rights. The motion was seconded and carried.

IV. RESIGNATION OF DR. HARRIS AS AMA ALTER-NATE DELEGATE

Dr. Harris' letter of resignation as AMA Alternate Delegate was read. Dr. Lang moved that Bruce Lushbough, M.D., be appointed to complete the term of AMA Alternate Delegate. The motion was seconded and carried.

V. BLUE SHIELD PARTICIPATING AGREEMENT FORM The Council reviewed the discussion from the Corporate Body meeting concerning the Blue Shield participating agreement form which is to become effective July 1, and the appointment of a subcommittee to study and make recommendations regarding this form. Dr. Ferrell asked that any physicians interested in serving on this subcommittee contact the executive office, that appointments would be made soon, and the committee could then meet with representatives of Blue Shield to consider the proposed form.

VI. DR. RIAL'S OFFICIAL FAMILY BRIEFING Dr. Rial briefly addressed the Council concerning the JCAH. Dr. Rial also remarked on the proposal to allow residents and students to be seated on the House of Delegates as voting members. Dr. Barrett presented a centennial belt buckle to Dr. Rial in appreciation of his visit to South

Dakota.

The meeting adjourned at 11:30 p.m.

REMARKS BY WILLIAM Y. RIAL, M.D. PRESIDENT, AMERICAN MEDICAL ASSOCIATION

Dr. Rial stated that physicians should actively become involved in changing the negative image that has been conveyed to the public. This negative image has developed as a result of some individuals using their medical degree as a means of "holding up" their patients. People are concerned about the cost of medical care, but it has never been determined how much is too much medical care and how much is too little. He recommended that the medical profession take the leadership and be responsible purchasing agents in order to alleviate this negative image of physicians.

Dr. Rial explained that when the Federal Trade Commission order came out, the AMA affiliated state and county medical societies could not under any circumstances interfere with the

contractual arrangements by which doctors get paid and that physicians were not allowed to proscribe in any way against advertising unless it was fraudulent or deceptive. He commented that many county medical societies have had to abandon their grievance committees because most of the complaints heard were against a physician who charged too much or charged inappropriately, and that amounts to price fixing, which is in strict violation of the Federal Trade Commission's edict against the medical profession. Dr. Rial suggested that non-physicians or lay people comprise at least one-third to one-half of the members of a grievance committee to avoid being accused by the Federal Trade Commission of price fixing if this committee tries to moderate or adjust fees.

Regarding the JCAH, Dr. Rial reported that the term "medical staff" would be reinstated in place of organized staff in the

Accreditation Manual for Hospitals.

MINUTES OF THE FIRST HOUSE OF DELEGATES MEETING

Friday June 3, 1983 Ramada Inn Sioux Falls, SD

The first meeting of the House of Delegates was called to order at 8:30 a.m. by A. J. Barrett, M.D., Speaker of the House. Present for roll call were the following members of the House: Drs. Durward Lang, J. N. Hamm, Howard Saylor, Jr., W. O. Rossing, A. J. Barrett, G. E. Tracy, Russell Harris, B. C. Lushbough, A. A. Lampert, Jr., David Buchanan, R. G. Gere, Guy Tam, Larry Sittner, Lowell Hyland, Michael Pekas, Richard Porter, Frank Messner, Roger Millea, Robert L. Ferrell, James Jackson, M. G. Thompson, Charles Pelton, James Larson, Parry Nelson, Charles S. Roberts, Richard Holm, John Davis, William G. M. Huet, Ravi Kapur, Walter Baas, T. J. Bhatti, V. Brandenburg, George Bruins, Michael Ferrell, David Ohrt, Ronald Wyatt, Gail Benson, Jeffrey Hagen, Jerry Freeman, Roger Stoltz, Jay Hubner, R. J. Foley, Michael McVay, Richard P. Renka, James Kullbom, N. R. Whitney, Robert Stiehl, and David Yecha. A quorum being present, the meeting was declared competent to proceed.

Dr. Harris moved that the reading of the minutes of the last meeting be dispensed with inasmuch as the minutes have been published in the South Dakota Journal of Medicine.

The motion was seconded and carried.

Dr. Barrett then introduced Marlys Porter, president of the S.D. Woman's Auxiliary who presented Mrs. Betty Payne, president of the AMA Auxiliary, who brought a brief message of greeting to the House of Delegates.

Dr. Barrett introduced Dr. Durward Lang, president of the South Dakota State Medical Association, who presented the

following awards:

C. B. Alford Award — Robert H. Quinn, M.D. Past President's Award — Bruce Lushbough, M.D. Community Service Award — T. H. Sattler, M.D. Distinguished Service Award — O. J. Mabee, M.D. Special Presidential Award — G. E. Tracy, M.D. 50 Year Awards — G. I. W. Cottam, M.D.

A. W. Spiry, M.D. J. A. Eckrich, Sr., M.D. Paul Tschetter, M.D.

Dr. Berglund, a member of the AMPAC Board presented an award to SoDaPAC, third place nationally for contributions per member.

Mrs. Kay Reaney, Auxiliary AMA-ERF chairman, presented a check to Robert H. Quinn, M.D., Dean of the USD School of Medicine, in the amount of \$21,456.04, representing money raised by the South Dakota Auxiliary for AMA-ERF.

Dr. Barrett then announced the appointments to the Nominating Committee which have been made by Dr. Lang, president.

District 1 — Susan Ostrowski, M.D. District 2 — James Larson, M.D.

District 3 — Arthur Lampert, Jr., M.D.

District 4 — John Davis, M.D.

District 5 — David Buchanan, M.D.

District 6 — Charles D. Monson, M.D.

District 7 — Lowell Hyland, M.D. District 8 — Frank Messner, M.D.

District 9 — Robert Ferrell, M.D., Chairman District 10 — M. George Thompson, D.O.

District 11 — David Yecha, M.D. District 12 — Eldon Bell, M.D.

Dr. Barrett then announced the appointments to the various reference committees which he, as Speaker of the House, has

Reference Committee on Credentials, Resolutions and Memorials, and Reports of Officers and Councilors

William G. M. Huet, M.D., Chairman

Robert Stiehl, M.D.

T. H. Bhatti, M.D.

Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations David Yecha, M.D., Chairman

Kenneth Halverson, M.D.

James Larson, M.D.

Reference Committee on Reports of Commissions on Scientific Medicine, Internal Affairs, Communications and Liaison, and Professional Liability

Jeffrey Hagen, M.D., Chairman

David Sandvik, M.D. Parry Nelson, M.D.

Reference Committee on Reports of Special Committees and Miscellaneous Business.

James Jackson, M.D., Chairman

Walter Baas, M.D.

Richard Holm, M.D.

Dr. Lang moved that the reports of the president, president elect, vice president, secretary-treasurer, chairman of the Council, delegate and alternate delegate to the AMA, speaker of the house, councilor at large, executive secretary and councilors not be read and that these reports be referred to the Reference Committee on Credentials, Resolutions and Memorials, and Reports of Officers and Councilors. The motion was seconded and carried.

Dr. Gere moved, that on recommendation from the Council, the House of Delegates request that the appropriate reference committee draft a resolution stating the purpose and objectives of the USD School of Medicine for consideration and action at the Second House of Delegates meeting.

Dr. Barrett referred the above action to the Reference Committee on Reports of Special Committees and Miscellaneous Business.

Dr. Barrett then made the following referrals of reports included in the Delegates Handbook. The reports on pages 1-14 were referred to the Reference Committee on Credentials, Resolutions and Memorials, and Reports of Officers and Councilors. The reports on pages 15-17 in the handbook are referred to the Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations. The reports on pages 18-22 in the handbook are referred to the Reference Committee on Reports of the Commissions on Scientific Medicine, Internal Affairs, Communications and Liaison, and Professional Liability. The reports on pages 23-25 are referred to the Reference Committee on Reports of Special Committees and Miscellaneous Business.

Resolution #1, submitted by the Watertown District Medical Society concerning SoDaPAC was referred to the Reference Committee on Reports of Special Committees and Miscellaneous Business.

RESOLUTION #1

TO: House of Deletates

> South Dakota State Medical Association Watertown District Medical Society

SUBJECT: SoDaPAC

FROM:

WHEREAS, physicians collectively became involved in government and candidate support by organizing the

South Dakota Physicians Committee in 1960; and

WHEREAS, the committee became the South Dakota Political Action Committee (SoDaPAC) in 1963; and

WHEREAS, SoDaPAC contributes funds to aid in the election of South Dakota candidates for the Legislature of South Dakota and congressional candidates whose philosophy is similar to the medical profession; and

WHEREAS, physicians control SoDaPAC and realize that financial contributions are essential to continuing the influential political voice of medicine in South

THEREFORE, BE IT RESOLVED that the South Dakota Medical Association reaffirm its support for SoDaPAC and encourage its members and their spouses to join and become active members.

Bylaw Revision #1, concerning subcommittees of commissions was referred to the Reference Committee on Reports of Special Committees and Miscellaneous Business.

BYLAW REVISION #1

TO: House of Delegates

South Dakota State Medical Association

FROM: The Council

South Dakota State Medical Association SUBJECT: Bylaw Change Regarding Subcommittees ARTICLE X

(((Section 6. Subcommittees

Any commission authorized by this article may appoint subcommittees to assist it in fulfilling its responsibilities under these Bylaws. Subcommittees shall be appointed by the commission of which the subcommittee is a part with the approval of the Council. At least one subcommittee member shall be a member of the commission of which the subcommittee is a part. Subcommittee members who are not commission members may vote on subcommittee business and reports, which must be approved by the commission, but they shall not be voting members of the commission, solely by reason of their membership on a subcom-

Dr. Barrett made several announcements concerning the events of the annual meeting. On motion, the meeting adjourned at 9:15 a.m.

MINUTES OF THE SECOND HOUSE OF **DELEGATES MEETING**

9:30 a.m. Washington Room, Ramada Inn Sioux Falls, South Dakota Sunday, June 5, 1983

The second meeting of the House of Delegates was called to order at 9:45 a.m. by Arthur J. Barrett, M.D., Speaker of the House. Present for roll call were the following members of the House: Drs. Durward Lang, Joseph N. Hamm, Howard L. Saylor, Jr., William O. Rossing, Arthur J. Barrett, Gerald Tracy, Bruce C. Lushbough, J. A. Eckrich, Jr., Arthur A. Lampert, Jr., David Buchanan, Richard G. Gere, R. E. Gunnarson, Guy Tam, Michael Pekas, Richard Porter, Frank Messner, Roger Millea, Robert L. Ferrell, James Jackson, M. George Thompson, James Wunder, James Larson, Parry Nelson, Charles S. Roberts, Richard Holm, William G. M. Huet, Ravi Kapur, Tajammul Bhatti, Verdayne Brandenburg, George Bruins, David Ohrt, Ronald Wyatt, Gail Benson, Jeffrey Hagen, Jerry Freeman, Roger Stoltz, Kenneth Halverson, Jay Hubner, R. J. Foley, Richard P. Renka, David Yecha, and Eldon Bell. A quorum being present, the meeting was declared competent to proceed.

Dr. Saylor moved that the reading of the minutes of the last meeting be dispensed with inasmuch as they will be published and distributed. The motion was seconded and carried.

Dr. Robert Ferrell read the report of the Nominating Commit-

REPORT OF THE NOMINATING COMMITTEE

The Nominating Committee submits the following recommendations for the consideration of the House of Delegates:

COUNCILORS — 3 year terms

Granville Steele, M.D. Aberdeen District #1 Watertown District #2 Pierre District #4 Sioux Falls District #7 Yankton District #8 Black Hills District #9 ALTERNATE COUNCILORS

James C. Larson, M.D. R. C. Jahraus, M.D. John Ochsner, M.D. Richard Tschetter, M.D. Frank Messner, M.D. James Jackson, M.D. 3 year terms Jay Bachmayer, M.D. G. Robert Bartron, M.D. M. R. Cosand, M.D. K. Gene Koob, M.D.

Daniel Kennelly, M.D.

Dr. Ferrell moved that nominations cease and a unanimous ballot be cast for the proposed slate of councilors and alternate councilors. The motion was seconded and carried. **OFFICERS**

President Elect

Aberdeen District #1 Watertown District #2

Pierre District #4 Sioux Falls District #7

Howard L. Saylor, Jr., M.D.

Dr. Hamm moved that nominations cease and a unanimous ballot be cast for Dr. Howard L. Saylor, Jr. as President Elect. The motion was seconded and carried. Richard G. Gere, M.D. Vice President

Dr. Rossing moved that nominations cease and a unanimous ballot be cast for Dr. Richard G. Gere as Vice Presi-

Speaker of the House

A. J. Barrett, M.D.

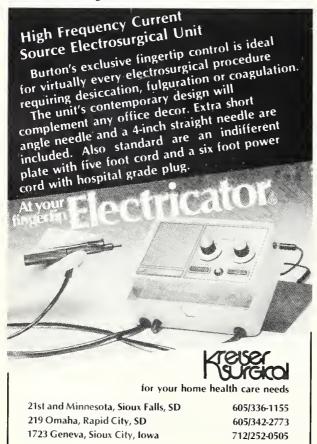
Dr. Saylor moved that nominations cease and a unanimous ballot be carried for A. J. Barrett as Speaker of the House. The motion was seconded and carried.

ANNUAL MEETING SITE

1984 — Rapid City, SD, June 7, 8, 9, 10, 1984 1985 — Sioux Falls, SD, June 6, 7, 8, 9, 1985

1986 — Rapid City, SD

The Nominating Committee recommends that the membership be surveyed to determine if Aberdeen should be added to the annual meeting rotation.



Dr. Lang moved that the House not accept the recommendation to survey the membership regarding future annual meeting sites. The motion was seconded and carried.

Respectfully submitted, NOMINATING COMMITTEE Robert Ferrell, M.D., Chairman Susan Ostrowski, M.D. James Larson, M.D. Arthur Lampert, Jr., M.D. John Davis, M.D. David Buchanan, M.D. Lowell Hyland, M.D. Frank Messner, M.D. M. George Thompson, D.O. David Yecha, M.D.

Dr. Lang moved to accept the report of the Nominating Committee as amended. The motion was seconded and carried.

Dr. Barrett introduced Dr. William Y. Rial, President of the AMA from Swarthmore, Pennsylvania. Dr. Rial briefly addressed the House regarding the dilemma concerning health care costs and quality of care, the Federal Trade Commission's attempt to control price fixing, and the JCAH.

Dr. Huet read the report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers

and Councilors.

REPORT OF THE REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND MEMORIALS AND REPORTS OF OFFICERS AND COUNCILORS

The following delegates, alternate delegates, officers and councilors of the South Dakota State Medical Association were present: Drs. Durward Lang, Joseph Hamm, Howard Saylor, Jr., William O. Rossing, Arthur J. Barrett, Gerald E. Tracy, Russell Harris, Bruce C. Lushbough, Arthur A. Lampert, Jr., David Buchanan, Richard Gere, Guy Tam, Larry Sittner, Lowell Hyland, Michael Pekas, Richard Porter, Frank Messner, Roger Millea, Robert L. Ferrell, James Jackson, M. George Thompson, James Wunder, Charles Pelton, Susan Ostrowski, James Larson, Parry Nelson, Charles S. Roberts, Richard Holm, John Davis, William G. M. Huet, Ravi Kapur, Walter Baas, T. Bhatti, V. Brandenburg, George Bruins, Michael Ferrell, David Ohrt, Ronald Wyatt, Gail Benson, Jeffrey Hagen, Jerry Freeman, Roger Stoltz, Edward Clark, Jay Hubner, R. J. Foley, Michael McVay, R. P. Renka, James Kullbom, N. R. Whitney, Robert Stiehl, and David Yecha.

A quorum was present for the meeting of the House of Delegates. Total registration for the convention is 435, including 273 physicians, 21 guests, 104 Auxiliary members and 37

The committee submits the following resolution for the con-

sideration of the House of Delegates:

WHEREAS, the Seventh District Medical Society, the Seventh District Auxiliary and the Third District Auxiliary members have been so thorough in making arrangements for the success of the combined meeting of our Annual Meeting,

BE IT RESOLVED, that the South Dakota State Medical Association give its voice in appreciation and thanks to the local physicians in the Seventh District and the members of the Seventh District Auxiliary and the Third District Auxiliary

WHEREAS, the management of the Ramada Inn has been so cooperative in providing facilities for the success of the Annual Meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Ramada Inn.

WHEREAS, The Sioux Falls Argus Leader, the Sioux Falls Tribune, KELO TV, KSFY TV, KELO radio, KSOO radio, KRSS radio, KKRC radio, KXRB radio and KNWC radio have been most cooperative in presenting the public news of the Annual Meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks to the Sioux Falls Argus Leader, the Sioux Falls Tribune, KELO TV, KSFY TV, KELO radio, KSOO radio, KRSS radio, KKRC radio, KXRB radio and KNWC

WHEREAS, the Minnehaha Country Club has been most cooperative in providing facilities for the golf tournament and Thursday evening stag party and the Auxiliary luncheon on Saturday,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the

Minnehaha Country Club.

BE IT RESOLVED, that \$50 be donated to the South Dakota Medical School Endowment Association in memory of the following physicians who died during the past year:

John F. Hill, M.D. F. J. Radusch, M.D. Frederick Rosenfeld, M.D. R. E. Lemley, M.D.

Inasmuch as the South Dakota School of Medicine, the South Dakota Medical School Endowment Association and many other medically related organizations are continuously in need of additional funds, the committee would like to encourage members of the South Dakota State Medical Association to remember these in their wills.

The committee reviewed the reports of the officers and councilors and recommends they be accepted as submitted.

The committee would like to recognize specifically the University of South Dakota School of Medicine, its work in supporting community and state awareness of medicine, continuing medical education, and in elevating quality of medical care. The committee encourages continued State Medical Association support for the school.

The committee would also like to recognize the superb and outstanding work and ability of our Executive Secretary, Robert Johnson, and his staff during the last year in conducting the business of the South Dakota State Medical Association.

Respectfully submitted,

REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND REPORTS OF OFFICERS AND COUNCILORS

William G. Huet, M.D., Chairman

Robert Stiehl, M.D. T. H. Bhatti, M.D.

Dr. Lang moved to accept the report of the Reference Committee on Credentials, Resolutions and Memorials and Report of Officers and Councilors. The motion was seconded and carried.

Dr. Yecha read the report of the Reference Committee on Reports of the Commissions on Medical Service; Legislation and Governmental Relations.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommended that legislation be introduced increasing the drinking age to 21 and recommended support of this legislation if introduced. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations with the comment above and would like to commend the Commission for their work during the year.

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service and would like to commend the Commission for their

work during the year.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

David Yecha, M.D., Chairman James Larson, M.D. Michael McVay, M.D., for

Kenneth Halverson, M.D.

Dr. Lang moved to amend the report to state as follows: "The Reference Committee recommended that legislation be considered increasing the drinking age to 21 and recommended support of this legislation if introduced." The amendment was seconded and carried.

Dr. Saylor moved to accept the report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations as amended. The motion was seconded and car-

Dr. Hagen read the report of the Reference Committee on Reports of the Commissions on Scientific Medicine; Internal Affairs, Communications and Liaison; and Professional Liabil-

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSIONS ON SCIENTIFIC MEDICINE, INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON, AND PROFESSIONAL LIABILITY

The Reference Committee reviewed the report of the Commission on Scientific Medicine. The Reference Committee supports the Committee on Long Range Planning in their survey of the membership regarding future annual meetings and hopes the information received will be of assistance to the Commission on Scientific Medicine in planning future meetings. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends acceptance of this report.

The Reference Committee wishes to commend the members of the three Commissions for their work on behalf of the members of the State Medical Association during the past year.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF THE COMMISSIONS ON SCIENTIFIC MEDICINE, INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON, AND PROFESSIONAL LIABILITY Jeffrey Hagen, M.D., Chairman Parry Nelson, M.D. David Sandvik, M.D.

Dr. Larson moved to accept the report of the Reference Committee on Reports of the Commissions on Scientific Medicine; Internal Affairs, Communications and Liaison; and Professional Liability. The motion was seconded and carried.

Dr. Jackson read the report of the Reference Committee on Reports of Special Committees and Miscellaneous Business.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

The Reference Committee has reviewed and recommends acceptance of reports from the Committee for Continuing Medical Education, the Long Range Planning Committee, the Archives and History Commission, the Liaison Committee, the South Dakota Political Action Committee, the Board of Directors of the South Dakota Medical School Endowment Association and the president of the South Dakota Medical School Endowment Association. The Reference Committee has reviewed and recommends acceptance of the report of the Grievance Commission reiterating that when a physician critizes a peer, "it is imperative that when this criticism is made, it is well documented and that all of the facts are known."

The Reference Committee has reviewed and recommends the acceptance of Resolution #1.

The Reference Committee has reviewed and recommends the acceptance of Bylaw Revision #1.

The Reference Committee submits and recommends acceptance of the following resolution:

RESOLUTION #2

WHEREAS, the original and primary mission of the University of South Dakota School of Medicine actually, historically and conceptually has been the accomplishment of quality medical student education and enhancement of high levels of primary and specialty medical care to the citizens of South Dakota, and

WHEREAS, medical research has not been actually, historically or conceptually a priority mission of the University of South Dakota School of Medicine, and

WHEREAS, the faculty of the University of South Dakota School of Medicine has been recruited and heretofore promoted and retained for the accomplishment of the above stated primary mission of the University of South Dakota School of Medicine, and

WHEREAS, recruitment, appropriate promotion and retention of quality volunteer and full time clinical and academic faculty is essential to the continued viability of the University of South Dakota School of Medicine, and

WHEREAS, recent promotion procedures may have changed in a fashion that may jeopardize the appropriate advancement, retention and recruitment of quality faculty,

NOW THEREFORE BE IT RESOLVED, that the South Dakota State Medical Association recognizes and reaffirms the primary mission of the School of Medicine as defined by the legislature and the State Medical Association and the University of South Dakota as education and service, and

BE IT FURTHER RESOLVED, that the South Dakota State Medical Association endorses promotion procedures which allow the appropriate advancement and recruitment of faculty in relation to the mission and purpose of the medical school, and

BE IT FURTHER RESOLVED, that the South Dakota State
Medical Association recognizes that medical research and promotion therefore, is a desirable
goal of the University of South Dakota School of
Medicine if the appropriate commitment to that
research is provided in terms of facilities, funding, time allotment and personnel.

Dr. Saylor moved to commend the Reference Committee for their excellent job in writing this resolution. The motion was seconded and carried.

Respectfully submitted, REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS James Jackson, M.D., Chairman Walter Baas, M.D. Richard Holm, M.D.

Dr. Buchanan moved to accept the report of the Reference Committee on Reports of Special Committees and Miscellaneous Business. The motion was seconded and carried.

Dr. Barrett administered the Oath of Office to Joseph N. Hamm, M.D., 1983-84 president. Dr. Hamm thanked the House for the confidence they have shown by electing him president and stated he will fulfill his responsibilities to the best of his ability.

Dr. Barrett introduced the new officers, councilors and alternate councilors to the House members. Mr. Johnson announced

the Council would meet immediately following adjournment of the House in the Roosevelt Room.

The meeting adjourned at 11 a.m.

REPORT OF THE PRESIDENT AND CHAIRMAN OF THE EXECUTIVE COMMISSION

As your president, it has been my pleasure to represent you at many levels. Your Medical Association is active in many areas which deserve mention.

On the national level, your AMA Delegate, Jerry Tracy; AMA Alternate Delegate, Russ Harris, Executive Secretary, Bob Johnson; and myself attended the AMA Annual Meeting and the Interim Meeting. At these meetings, we also represented you at the North Centeral Conference, where we cooperated with Nebraska, Iowa, North Dakota, Minnesota and Wisconsin in areas of mutual concern.

At the state government level, we met with Governor Janklow and his staff and the South Dakota Hospital Association to try to find some realistic ways to reduce Medicaid costs. This was necessitated by excessive budget over-runs at the state level. You saw some of the results in the legislative session in Pierre. We found Governor Janklow to be very cooperative and appreciative of efforts to hold down costs.

The legislative session was a qualified success. The legislature has requested a thorough study of health care costs, the nature of which may take almost any direction.

Your Executive Secretary will coordinate our efforts to bring out the facts regarding health care costs in South Dakota in conjunction with the Executive Commission.

All items to come before the Executive Commission were subsequently discussed or previously discussed by the Council.

The staff of the South Dakota Medical Association deserves special mention. They are an able, talented group with the best interests of our Association firmly in mind. They have been invaluable to all of us.

Respectfully submitted,

Respectfully submitted, Durward Lang, M.D. President and Chairman Executive Commission

The Reference Committee reviewed the report of the President and Chairman of the Executive Commission and recommends it be accepted as submitted.

REPORT OF THE PRESIDENT-ELECT

The President-Elect has attended meetings of the House of Delegates, the Council, the Executive Commission and the AMA Leadership Conference which convened in Chicago in January of 1983. Working with the members, officers and staff has been most rewarding. I am humbly grateful that the membership has given me an opportunity to serve as your President for the coming year. I look forward to that assignment.

Respectfully submitted, Joseph N. Hamm, M.D. President-Elect

The Reference Committee reviewed the report of the President-Elect and recommends it be accepted as submitted.

REPORT OF THE VICE PRESIDENT

As Vice President of the South Dakota State Medical Association, I have attended the meetings of the Council and Executive Commission of the Medical Association. The very active participation of all members of the Council, Commission Chairmen and members of the Commission are essential for the continued progress in providing the top-notch medical care that we are so proud of in South Dakota. I am sure that this will continue on an ascending scale, and I would like you to know that I am always available for any help that I may provide for problems that have to do with delivery of health care in South Dakota.

Respectfully submitted, H. L. Saylor, Jr., M.D. Vice President

The Reference Committee reviewed the report of the Vice President and recommends it be accepted as submitted.

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REPORT OF THE SECRETARY-TREASURER

The past year has seen continued growth in membership of the South Dakota State Medical Association due to increasing numbers of physicians entering the state and greater interest on the part of the existing population in medical affairs.

As noted by the budgetary report, our financial condition continues to improve as a result of a larger dues base and prudent investment decisions made under the guidance of our Executive

Secretary.

During the year, I have attended meetings of the Council and the Executive Committee on a regular basis, along with special

meetings called by the President.

The Secretary-Treasurer also serves as an appointed member of the Health Professions Loan Advisory Board of the State of South Dakota; however, during the calendar year 1982 no meetings were convened by this group.

Respectfully submitted, W. O. Rossing, M.D. Secretary-Treasurer

The Reference Committee reviewed the report of the Secretary-Treasurer and recommends it be accepted as submitted.

REPORT OF THE CHAIRMAN OF THE COUNCIL

The Council of the South Dakota State Medical Association met at its usual times. The matters to come before the Council continue to be many and varied as the number of physicians in the state increase. Each district has seen to it that able Councilors are elected, thus making the Council function well.

Again, one should also mention the work of the Commissions and the Commission Chairmen, who do a great deal of the work

prior to the matters coming to the Council.

I would like to specifically thank our Executive Secretary, Mr. Robert Johnson, and his very able, capable staff for all the work that they put in to make the Council function smoothly.

Respectfully submitted, Richard G. Gere, M.D. Chairman, Council

The Reference Committee reviewed the report of the Chairman of the Council and recommended it be accepted as submitted.

REPORT OF THE AMA DELEGATE

It has been my pleasure to represent South Dakota at both the interim meeting and the annual meeting in 1982. Dr. Russell Harris, the alternate AMA delegate, and Dr. Durward Lang, State Association president, also attended both AMA meetings along with Robert Johnson, executive secretary. A report on AMA actions is sent to all members following each AMA meeting for your information.

This delegate continues to be impressed with the role of even the small states in the total organization of the AMA. I believe the AMA is addressing areas of concern to practicing physicians in smaller rural areas, and would encourage all South Dakota doctors to become members and participate in organized medicine at the national level. It has been my pleasure to serve as South Dakota's representative to the AMA, and I look forward to attending the 1983 sessions as your Delegate.

Respectfully submitted, Gerald E. Tracy, M.D. AMA Delegate

The Reference Committee reciewed the report of the AMA Delegate and recommended it be accepted as submitted.

REPORT OF THE AMA ALTERNATE DELEGATE

As the AMA Alternate Delegate, I have attended meetings of the Council and Executive Commission during the past year. I also attended both the interim and annual sessions of the AMA during 1982. The Delegate and I report the actions of each House session to the membership after the meetings, and we hope these reports are of value to you.

I believe the American Medical Association is an extremely

important segment of organized medicine, and I would encourage all physicians to join and become active participants.

Respectfully submitted, Russell H. Harris, M.D. AMA Alternate Delegate

The Reference Committee reviewed the report of the AMA Alternate Delegate and recommended it be accepted as submitted.

REPORT OF THE SPEAKER OF THE HOUSE

I look forward to presiding at the upcoming meetings of the House of Delegates in Sioux Falls. I thank my colleagues who have accepted appointments to the reference committees and particularly those serving as chairmen. The active participation of the reference committees and the involvement of the entire House of Delegates makes the deliberations and decisions of the State Medical Association more expedient and truly representative of our membership.

As Speaker of the House, I have attended two Council meetings and participated in deliberations of the Executive Commis-

sion.

Respectfully submitted, Arthur J. Barrett, M.D. Speaker of the House

The Reference Committee reviewed the report of the Speaker of the House and recommended it be accepted as submitted.

REPORT OF THE COUNCILOR AT LARGE

I have been privileged to serve as Councilor at Large for the past year and have attended most of the council meetings and executive committee meetings during that time.

It has been a pleasure to serve in your behalf as Councilor at Large during the past year.

Respectfully submitted, B. C. Lushbough, M.D. Councilor at Large

The Reference Committee reviewed the report of the Councilor at Large and recommended it be accepted as submitted.

REPORT OF THE EXECUTIVE SECRETARY

Throughout this past year, Dr. Lang and I have enjoyed visiting each of the twelve district medical societies. It is always a privilege to see my old friends and make new acquaintances during these visits. The hospitality shown us in 1982-1983 was gratefully appreciated and we left each meeting with the feeling of unity within medicine. It is often said that grass roots unity of any association is the determining factor in that association's survival. It is comforting to know and see firsthand as we make our district visits that each district grows, not only in terms of professional growth, but in community activity as well. The leadership demonstrated by your district officers assists all of us in achieving this desired objective. In conversations with many Association members, they have expressed great satisfaction with the productive meetings their district officers have scheduled. I applaud and encourage each of you to continue active participation in district activities.

The reports of the standing Commissions of the State Medical Association are included elsewhere in your handbook and I will not reiterate points in their reports; however, I think it is only appropriate to extend a heartfelt "thank you" to all commission and committee chairmen and members. Also, I would like to thank the many physicians who serve on committees of other organizations. Through their participation on the SoDaPAC Board of Directors, the Board of Medical and Osteopathic Examiners, the several Health Planning Agencies, the Endowment Association Board, and others, the Association benefits by increased public awareness of medicine's commitment to the many varying programs and projects which benefit our society.

The 1983 legislative session commenced at a slow and noncontroversial pace; however, it soom became apparent that pace was to be short lived. Over thirty bills affecting the medical

profession in the state of South Dakota were introduced. In addition, other bills were submitted which, had they been amended, would have impacted your practice. Although Dr. Haas, Chairman of the Commission on Legislation and Governmental Relations has summarized the Commission's final legislative program in his report, there are several matters which I would like to address briefly. The State Department of Health again introduced legislation to extend Certificate of Need to physician's offices (HB 1244) when major medical equipment valued at or in excess of \$400,000 is purchased. The bill was successful. I am convinced that although a small minority of legislators favor the concept contained in Certificate of Need, the legislature would have tabled the bill had not the federal government threatened the state with the potential loss of several million dollars of federal matching money. We were able to amend the bill to require a termination or suicide clause of the provision applicable to physician's offices should federal requirements be deleted by Congress. Another example of legislation expanding government regulation or control over the private sector was SB 188, a bill creating a State Office of Laboratory Services. The bill would have vested control in the Director of Laboratories to oversee the purchase and assignment of all laboratory equipment costing in excess of \$1,000, and other broad and far-reaching powers. The contention of the bill's sponsor was that it would save the state money by centralizing laboratory services. It seems to us that to truly save money, all testing should be performed by the private sector through a competitive bidding process. Fortunately, this bill was defeated, but there is reason to believe that this bill, or one similar, will surface again next year. I would be remiss if I failed to mention Senate Concurrent Resolution #30. SCR30 requests a summer study of health care costs by the Legislative Research Council Executive Board. This Board will sclect legislators to study hospital discount plans, hospital reimbursement practices and costs, the relationship of health care providers, health insurers and private parties, and assigned risk health care insurance. The issue of state Medicaid dollars and how to decrease the state's cost while at the same time maintaining current levels of services will no doubt be with us in the months ahead.

At the national level, a major disappointment to organized medicine is the still unanswered question of Federal Trade Commission jurisdiction over the learned professions and their organizations. Although legislation favorable to us was passed out of the House of Representatives, similar amended legislation failed on a 14-15 votc in the Senate Appropriations Committee, of which Senator James Abdnor is a member. The Senate passed the bill containing the offensive Rudman amendment, as sent out by the Committee. As a result of conflicting language between the House and Senate versions, a conference committee was appointed. They struck the objectionable language as passed by the full Senate, and as a result, the law remains as it always has been . . . unclear. The battle goes on. Another heated topic has been the dramatic change to the Medicare program which was passed by the House of Representatives on March 9. As of the writing of this report, the Senate has not yet taken action. The change entails the payment to hospitals on the basis of 467 diagnosis related groups (DRG's) regardless of the cost actually incurred in treating patients. The bill also requests that the Sccretary of Health and Human Services report to Congress by December 31, 1984, on "the advisability and feasibility of making physician payments under a prospective payment system." One further example of the strong challenge facing medicinc is a proposed regulation by the Health Care Financing Administration. The regulation would, if adopted, impose minimum conditions on hospitals participating in Medicare and Medicaid and who are not accredited by JCAH. Within this regulation, the term "physician" is defined to include dentists, podiatrists, optometrists, and chiropractors in addition to doctors of medicinc and ostcopathy. The above changes by Congress, both actual and proposed, place organized medicine in a precarious position. We must continue to monitor legislation as it affects medicine and promote legislation beneficial for the medical profession.

It has been said that "political responsibility is responsible medicine." I firmly believe that all physicians and their spouses should be at least, regular members of SoDaPAC in both election and off-election years. If you believe that a voice of one, whispers... a voice of many, influences, than I encourage you to join your colleagues who are SoDaPAC members. The time is now for all of us to become active participants in organized medicine's fight to maintain quality medical care at a reasonable cost. The battle lines are being drawn and the issues more distinct. Ask your colleagues in Iowa what has happened to medicine this past year.

For the sixth consecutive year, I am pleased to say that no dues increase is necessary this year, and that your Association is financially sound. Dr. Rossing, your Secretary/Treasurer, the Budget and Audit Committee and the Executive Commission have, as always, demonstrated foresight and guidance in setting financial policy beneficial to your Association. In addition, I would like to extend my grateful appreciation and commend your officers and councilors for their guidance in determining medicine's role in South Dakota. It has been the genuine pleasure of your staff to work with such highly motivated and dedicated physicians.

To your President and my dear friend, Dr. Lang, I extend our sincere thanks and commendation for truly representing the medical profession with honor and distinction. You have made us all proud to be associated with the greatest profession in the world. Even though many decisions which confronted you as president were difficult, the talent, dedication and leadership you provided were most beneficial to your colleagues and to the people of South Dakota.

Respectfully submitted, Robert D. Johnson Executive Secretary

The Reference Committee reviewed the report of the Executive Secretary. The Committee recognized the superb and outstanding work and ability of our Executive Secretary, Robert Johnson, and his staff during the last year in conducting the business of the South Dakota State Medical Association.



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REPORT OF THE FIRST DISTRICT COUNCILOR

The First District Medical Society continued to hold monthly meetings September through May, with the exception of January. The September meeting was featured as a presentation by Dr. Juan Munoz on "Insulin Receptors and Type II Diabetes." There was a discussion on concern about the itinerant practice of medicine in this area.

The October 6 meeting featured a presentation by Dr. B. Levine, a Cardiologist from Minneapolis, concerning congestive heart failure. Drs. James Alexander, Phyllis Heinemann and Warren Redmond were accepted for membership in the district society.

The November 3, 1982, meeting featured the president of the South Dakota State Medical Association, Dr. Durward Lang, and our Executive Secretary, Mr. Robert Johnson.

The December meeting featured entertainment by "The Black Hills Estrogenics." New officers elected were:

President: Dr. Jay Bachmayer
Vice-President: Dr. Harvey Hart
Secretary: Dr. Robert Brown
Delegates: Dr. Juan Chavier
Dr. Charles Pelton
Dr. David Seaman
Alternate Delegates: Dr. Susan Ostrowski

es: Dr. Susan Ostrowsk
Dr. Sterling Berg
Dr. Robert Brown

A donation of \$150 was made to the Women's Auxiliary for the AMA-ERF in appreciation of the entertainment.

The February 2, 1983, meeting featured a presentation by Dr. David Jagelman of the Cleveland Clinic on surgical aspects of

inflammatory bowel disorders.

The March 2, 1983, meeting featured a presentation by Dr. W. R. Palmer of Omaha, Nebraska, on treatment of rheumatoid arthritis, especially with the use of antiinflammatory agents. Dr. G. Steele was nominated as new Councilor for our district and Dr. J. Bachmayer was nominated as Alternate Councilor.

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SOUTH DAKOTA JOURNAL OF MEDICINE 608 West Avenue, North Sioux Falls, SD 57104 The April 13 meeting featured a presentation by Representative Joe Barnett who spoke on medical-legal issues and the local political scene.

The May 4 meeting featured Dr. Bruce Schilling who spoke on cranio-facial surgery.

Respectfully submitted, B. C. Gerber, M.D. J. A. Eckrich, Jr., M.D. Councilors, First District

The Reference Committee reviewed the report of the Councilors from the First District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SECOND DISTRICT COUNCILOR

Officers elected for 1983 for the Watertown District Medical Society include:

Dr. Richard McClaflin President Vice President Dr. James Horning Secretary-Treasurer Dr. Gerald Tracy Dr. James Larson (his alternate Delegate for 2 years is Dr. W. Nicol Guddal) Delegate for 1 year Dr. Parry Nelson (his alternate is Dr. James Horning) Dr. Theodore Wrage Censor for 3 years Censor for 2 years Dr. David Piro Censor for 1 year Dr. Bernie Hanson

Regular monthly meetings were held since the last annual meeting including business and scientific sessions. Dr. Durward Lang made his presidential visit to the district in October accompanied by the executive secretary, Robert Johnson. The overall activity of the Watertown District Medical Society has been excellent.

Respectfully submitted, G. Robert Bartron, M.D. Councilor, Second District

The Reference Committee reviewed the report of the Councilor from the Second District Medical Society and recommended it be accepted as submitted.

REPORT OF THE THIRD DISTRICT COUNCILOR

The Third Medical District of the South Dakota Medical Association is comprised of physicians in the towns of Brookings, Madison, Flandreau, Arlington, Estelline and Lake Preston. Meetings are scheduled for the group on an average of every two to three months. During the past year however, we were only able to meet four times because of weather conditions and other "emergencies." MEETINGS:

April 15, 1982:

The meeting was held in Brookings, South Dakota. The principal order of business at that time was discussion re-

June 17, 1982: garding the forthcoming State Meeting.
The meeting was held in Madison, South
Dakota. Dr. John Gregg from the South

Dakota State Health Department was originally scheduled to speak but had to cancel at the last minute. No formal education session was held but a routine business meeting was conducted.

December 9, 1982: The meeting was held in Flandreau, South Dakota. South Dakota Medical

Association President, Durward Lang, spoke regarding the South Dakota State Medical Association and its present role in health involvement in our state and

nationally.

February 24, 1983: The meeting was held in Madison, South

Dakota. The primary purpose for this meeting was to get all members of the District together including some new members and prospective members so we could visit and enjoy each others company.

April 14, 1983: The meeting was held in Brookings, South Dakota. An educational session was held with a speaker on sleep disorders.

New members to the District for the year 1983 include: Tad Jacobs, D.O., Flandreau; Merritt Warren, M.D., Brookings; K. Yemmanur, M.D., Estelline; S. Yemmanur, M.D., Estel-

Respectfully submitted, Arthur A. Lampert, M.D. Councilor, Third District

The Reference Committee reviewed the report of the Councilor from the Third District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FOURTH DISTRICT COUNCILOR

The Fourth District Medical Society met on January 12, 1983, for the official visitation by Dr. Durward Lang. He was accompanied by Robert Johnson and Kevin Loge. The program consisted of the presidential report and discussion of the upcoming legislative session.

The entire district participated in junior and high school athletic physical examinations for both Pierre and Fort Pierre in two separate sessions.

The following is a list of CME programs which were sponsored in conjunction with the Inservice Education Department of

St. Mary's Hospital: January 19, 1982 "Cutaneous Malignant Melanoma," Dr. D. W. Ohrt, L.C.M., Sioux Falls, SD, A.A.F.P., 2 hours P Teleconference, "Antibiotic Induced January 26, 1982

Colitis," Dr. Raszkowski, U.A.D., A.M.A.I., 1 hour

February 16, 1982 "Death Conference," St. Mary's Medical Staff

February 22-23, 1982 "CPR Certification"

"Developments in Peripheral Vascular March 16, 1982 Dr. Mattson, A.A.F.P., 2 Disease, hours P

"Evaluation and Treatment of Allergies April 20, 1982 in Children," Dr. Lowell Hyland, A.A.F.P., 2 hours P

"Apnea in the Newborn," Dr. Larry April 30, 1982 Fenton, A.A.F.P., 1 hour P "Clinical Diets," Debra Brakke,

May 18, 1982

A.A.F.P., 2 hours P July 8, 15, 1982 Teleconference, "Bronchopulmonary

Dysplasia

"Antibiotic Review," Dr. Donald September 21, 1982 Humphreys, A.A.F.P., 2 hours P

November 30, 1982 "Update on Preeclampsia," Dr. Dean Madison, A.A.F.P., 2 hours P

Respectfully submitted, R. C. Jahraus, M.D., Councilor, Fourth District

The Reference Committee reviewed the report of the Councilor from the Fourth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FIFTH DISTRICT COUNCILOR

The Huron District Medical Society has met regularly during the past year with both business and scientific sessions. During this year our membership has increased one, to 29 active members. Dr. R. J. Pelegrin discontinued his membership when he left South Dakota, and we gained two new members, Dr. Cynthia Kortum and Dr. Mark Belyea.

The officers for 1983 are William G. M. Huet, M.D., president; Ravi Kapur, M.D., vice president and Emil Hofer, M.D., secretary-treasurer. Delegates selected to attend the annual meeting are Dr. William Huet and Dr. Ravi Kapur; alternate delegates are Dr. Emil Hofer and Dr. Roscoe Dean.

Respectfully submitted, David Buchanan, M.D. Councilor, Fifth District

The Reference Committee reviewed the report of the Councilor from the Fifth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SIXTH DISTRICT COUNCILOR

The Sixth District of the South Dakota State Medical Association met on a much more regular basis this year than it has in the past. In addition to just a social evening, a scientific program was planned for a majority of them. Our district has now grown to 42 physicians with the prospect of further additions in the near future.

Officers for the year were President, C. D. Monson, M.D.; Vice President, John Jones, M.D.; Secretary-Treasurer, Tim Judge, M.D.; Delegates, W. P. Baas, M.D., and C. D. Monson, M.D.; Alternates were Maynard Porter, M.D., and Tim Judge,

It is hoped that we can continue to improve the quality of our meetings and the attendance.

> Respectfully submitted, R. G. Gere, M.D. Councilor, Sixth District

The Reference Committee reviewed the report from the Councilor for the Sixth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SEVENTH DISTRICT COUNCILOR

The following is the report as Councilor of the Seventh District Medical Society of the State Medical Association of South Dakota. The year 1982 was a moderately prosperous year for the Seventh District Medical Society. We accepted 28 new members into the Medical Society and three transfers in comparison to 1981 during which time 23 new members were accepted into the Society.

The January meeting was marked by the change of presidency, from Dr. Larry Finney to Dr. John Ochsner, and the program for that month was Medicare Reimbursement.

The following several months consisted of reports by Dr. Cloar concerning the status of his Family Practice Residency Program and the City Health Department of Sioux Falls. Reports were also given by Mr. Henry Morris and Mr. Jon Soderholm of the two hospitals in Sioux Falls concerning examples of daily hospital charges, breakdown as sources of income, projected needs, comparison of health costs in South Dakota as opposed to that of a national level. Dr. Warren L. Opheim was then elevated to honorary membership in the Seventh District Medical Socie-

The April meeting was marked by being celebrated as the 100th April meeting of the Seventh District Medical Society. Various topics of discussion by the Society during the several following months were the concerns of the Seventh District regarding Medicare reimbursement in general, the coroner's legislation then pending before the legislature, and the current status of the Lion's Eye Bank. The Medical Society went on record as endorsing the Lion's Eye Bank.

The May meeting was marked by Dr. Earl Kemp describing the patient education project undertaken by the Family Practice Center on KSFY's 6:00 p.m. news. This meeting was also marked by the discussion of the U.S. Department of Health and Human Services stand on mandatory prior notification of parents of teenagers when these teenagers ask for help in contraception. The District in general was in opposition to this proposal and supported the AMA's stand against this regulation.

Following the summer break, in September the following were welcomed into the Seventh District Medical Society as honorary members: Dr. T. J. Billion, Dr. John Dickinson and Dr. Charley McDonald. During the September meeting, review was made of the pending legislation clarifying the role of the

Federal Trade Commission with specific attention to bills HR3722, HR3995 and S2499. The Seventh District Medical Society pledged its support to the passage of these bills which are supported by the AMA. The program consisted of a panel discussion in the development of VA Hospital services by Mr. Dix and Drs. Assimacopoulos, Richards, Salem, Simmons and Quinn.

The October meeting was the annual bash at the Hiawatha Golf Club in Canton, South Dakota, and Dr. Quinn discussed the status of the Medical School including the recommendation of the recent LCME visit. As officers for 1983, Dr. Jerry Freeman was elected as treasurer, Dr. Jeffrey Hagen as secretary, and Dr. Rodney Parry as president elect. Delegates for a two year term including 1983 and 1984 to the South Dakota State Medical Association annual meeting are Drs. Tajammul Bhatti, Verdayne Brandenburg, George Bruins, Michael Ferrell, Donald Frost, David Ohrt, and Ronald Wyatt. Alternate delegates are Drs. Edward Clark, Vincent Cutshall, Roger Staltz, Daniel Kennelly, Dean Madison, Robert Marschke, Chuck Mohler, Jim Quale and Michael Stassen. Dr. Durward Lang then presented the annual South Dakota State Medical Association presidential address followed by a legislative update by Robert Johnson.

The December meeting was the annual meeting with wives and legislators.

Respectfully submitted, Larry L. Sittner, M.D. Councilor, Seventh District

The Reference Committee reviewed the report of the Seventh District Medical Society and recommended it be accepted as submitted.

BEING A PHYSICIAN AND A FAMILY MAN IS LIKE MAKING AN INCISION WITH A PARING KNIFE...

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REPORT OF THE EIGHTH DISTRICT COUNCILOR

The Eighth District Medical Society met four times during 1982 and 1983.

The first meeting was held Wednesday, April 28, 1982. John Sternquist was elected president of the Eighth District Medical Society. Dr. Bruce Lushbough, president of the State Medical Society, was present and addressed the group on multiple topics relevant to medicine and answered many questions from the physicians present. Bob Johnson, executive secretary, was also present and gave a short address and answered several questions. The Bylaws Committee, composed of Dr. Messner and Dr. Reaney, proposed several changes to the Bylaws. These were read and will be voted on at the next meeting. Dr. Chester McVay was voted to be made a life member of the Eighth District Medical Society and a resolution will be sent to the State Medical Association for a life membership.

The second meeting was held September 9, 1982. The previously noted Bylaw changes were again discussed and it was felt some additional changes should be made and these will be brought up in the future. Due to the fact that the Eighth District Medical Society had some extra money on hand, it was felt that a donation of \$1,500 could be made to the USD Medical School Endowment Fund. This was voted on and passed. Dr. Messner gave a talk dealing with the new CT scanner at Sacred Heart and demonstrated the capabilities in several interesting cases.

The third meeting was held December 1, 1982. The Bylaw changes were re-discussed, voted on, and passed. Dr. Durward Lang, President of the South Dakota Medical Association, met with the staff and gave a talk along with Bob Johnson. They both answered questions by the physicians. Dr. Messner reported on the medical school and the task force committee which had recently met.

Dr. Sattler was nominated for the Community Service Award. This motion passed. Drs. Pesce, Robison, Meyer, Provow, and Amyx were submitted for membership. They were approved. A 50-year plaque and pin were given to Dr. Hill. This was to be delivered to Dr. Hill's wife as he has recently passed away.

The fourth meeting was held February 23, 1983. A short business meeting was held with no significant business being discussed. An outside speaker was present and gave an extensive discussion on hemological abnormalities.

Respectfully submitted, F. D. Messner, M.D. Councilor, Eighth District

The Reference Committee reviewed the report of the Eighth District Medical Society and recommended it be accepted as submitted

REPORT OF THE NINTH DISTRICT COUNCILORS

During the past year, the Black Hills District Medical Society has met six times; May 11, 1982; September 9, 1982; November 9, 1982; December 9, 1982; February 1, 1983; April 14, 1983; and a seventh meeting was scheduled for May 10, 1983. At the September meeting, Dr. Durward Lang made his presidential visit accompanied by Robert Johnson, the executive secretary.

District membership for 1982 totaled 118 active members and 11 honorary members. District officers elected for 1983 include James Kullbom, M.D., president; Richard Renka, M.D., vice president; and Arthur J. Barrett, M.D., secretary-treasurer.

Respectfully submitted, Robert Ferrell, M.D. Roger Millea, M.D. James Jackson, M.D. Councilors, Ninth District

The Reference Committee reviewed the report of the Councilors from the Ninth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE TENTH DISTRICT COUNCILOR

The Rosebud District Medical Society has nine active members including Dr. Clark Marquart of Rosebud who joined during this past year. The district met in January at which time

Dr. Durward Lang made his presidential visit to the district along with Robert Johnson, executive secretary of the State

Officers elected for 1983 include:

Louis Hogrefe, M.D. President Delegate Robert Stiehl, M.D. R. G. Nemer, M.D. Alternate Delegate Respectfully submitted, M. George Thompson, D.O. Councilor, Tenth District

The Reference Committee reviewed the report of the Councilor from the Tenth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE ELEVENTH DISTRICT COUNCILOR

During the past year, eight meetings of the Eleventh District Medical Society were held. At these meetings, business sessions were conducted as needed. At seven of the meetings, scientific sessions were presented by visiting consultants in various specialties covering the topics as listed below:

April, 1982

Dr. Stephen Slaughterbech, dermatologist from Bismarck, North Dakota, spoke on "Dermatological Problems."

June, 1982

Dr. Joseph Kizer and Dr. Robert Van Tassel from Minneapolis, Minnesota, spoke on "Cardiovascular Aspects of Coronary Artery Disease" including percutaneous transluminal coronary artery dilations.

August, 1982

Dr. Allan Morris, University of South Dakota Medical School, spoke on "Update and Treatment of Arthritis.

September, 1982

Curt Wischmeier of Ellingson Eye Clin-

ic of Bismarck, North Dakota, spoke on "Eye Changes in Diabetes Mellitus,

Laser Treatment, and Vitrectomy.

Dr. Nick Neuman from the Heart and Lung Clinic of St. Alexius Hospital in Bismarck spoke on "The Care of the

Chronic Lung Patient and Pulmonary Rehabilitation.

January, 1983 Dr. Stanley Deide spoke on "What's New in the Treatment of Myocardial In-

farction.

February, 1983 Dr. Phillip Hoffsten spoke on "Evaluation and Treatment of Borderline

Hypertensive Patients.'

Our annual meeting was held in January, 1983, with Dr. Durward Lang, President, and Robert Johnson, Executive Secretary of the South Dakota State Medical Association, present to discuss legislative matters before the South Dakota Legisl-

New officers for the Society were elected in March of 1983 and are as follows:

President Vice President Secretary/Treasurer Delegate Alternate Delegate

November, 1982

Councilor

James D. Collins, M.D. Jeffrey L. Peterson, M.D. Leonard M. Linde, M.D. David Yecha, M.D. James D. Collins, M.D. James F. Wunder, M.D.

Respectfully submitted, James F. Wunder, M.D. Councilor, Eleventh District

The Reference Committee reviewed the report of the Councilor from the Eleventh District Medical Society and recommended it be accepted as submitted.

South Dakota Society Of **Pathologists**

Officers for 1982-83

Jerry L. Simmons, M.D., President Thomas E. Henry, M.D., Vice President Beth L. Johnson, M.D., Secretary-Treasurer



REPORT OF THE TWELFTH DISTRICT COUNCILOR

During 1982-83, the Whetstone Valley District Medical Soci-

ety met three times.

The first meeting was held in August in Rosholt at which time Dr. Lang made his official presidential visit. Bob Johnson, the Executive Secretary, also attended this meeting, and he and Dr. Lang discussed current topics of concern to the practicing physicians.

The district met in Milbank in December and in Webster in March. Both of these meetings included scientific speakers as well as business sessions. Officers elected for 1983 include Joseph Kass, M.D., President, and David Oey, M.D., Secretary.

Respectfully submitted, Joseph Kass, M.D. Councilor, Twelfth District

The Reference Committee reviewed the report of the Councilor from the Twelfth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The Commission on Legislation and Governmental Relations met only once during the year 1982-83. The meeting was held on

Friday, September 24, 1982.

The Commission met with Brad Randall, M.D., who reviewed in detail the draft legislation as passed by the House of Delegates in which various changes were proposed to the coroner's statutes. Dr. Randall also updated the Commission on the various interest groups in South Dakota which supported the changes. After further discussion, it was recommended that the Council and State Medical Association support the legislation.

A response from the South Dakota High School Activities Association to the State Medical Association's recommendation on changes for student athletic physical exams was given by Bob Johnson. The recommendation is still under further study and has been referred to the athletic directors of the state for their input. The Commission is to be kept informed as to the progress

or lack of progress on this matter.

Punitive damage coverage under state liability policies was discussed by the Commission. The first proposed piece of legislation would have required recovery in a civil action of double costs of defense of a punitive damage claim where the claim was found to be without reasonable basis. The second piece of legislation would have bifurcated the trial of negligence and trial for punitive damages. The Commission recommended that the State Medical Association sponsor the two legislative drafts for introduction in the 1983 South Dakota Legislature.

Information on licensure legislation to be introduced in the 1983 Legislature by the South Dakota Athletic Trainers Association was presented to the Commission. However, the Commission deferred action until specific legislation could be obtained.

The Commission reviewed proposed legislation which would have allowed a physician to treat an unemancipated minor without prior consent of a parent or legal guardian when, in the physician's judgement, an attempt to secure consent would increase the risk to the minor's life or health. Following discussion, the Commission recommended that the State Medical Association introduce the legislation.

The Commission reviewed a letter which had been submitted to all State Medical Associations and which addressed a proposal that would provide federal tax incentives to physicians who render assistance to the indigent. It was recommended that the Council accept the concept and that the State Medical Associa-

tion inform the AMA of this action.

A resolution from the Yankton District Medical Society as adopted by the SDSMA House of Delegates was reviewed by the Commission. Following lengthy discussion, the Commission recommended that the State Medical Association support legislation to strengthen South Dakota DWI statutes, that the State Medical Association not introduce legislation to increase the drinking age but provide educational assistance if such legislation was introduced, and that the State Medical Association

support the concept of child mandatory restraint systems if legislation was introduced.

Bob Johnson discussed with the members of the Commission the South Dakota Joint Appropriations Committee meeting on funding and utilization of Title XIX inpatient hospital care. Following discussion, it was moved that the State Medical Association support the imposition of deductibles or copayment as a cost containment mechanism in dealing with the excessive use and nonpayment of services.

The issue of Federal Trade Commission jurisdiction over the professions and the current status of congressional bills prohibiting such jurisdiction was discussed by the Commission. No action was taken as this was accepted for information only.

The rise in malpractice suits nationwide was discussed by the Commission and was accepted for information only.

Following is the finalized 1983 legislative program as adopted by the Commission.

SPONSORED BILLS

- Legislation creating an office of state coroner and amending present coroner's statutes.
- Recovery of double costs of defense of a punitive damage claim.
- 3. Bifurcation of trials of negligence and punitive damage.
- 4. Treatment of minors without prior parental consent.

ENDORSED BILLS

- 1. Legislation to strengthen DWI statutes.
- 2. Child mandatory restraint systems.

Respectfully submitted,
Stephen N. Haas, M.D., Chairman
Commission on Legislation
and Governmental Relations

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommended that legislation be introduced increasing the drinking age to 21 and recommended support of this legislation if introduced. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations with the above comment and would like to commend the Commission for their work during the year. The House of Delegates amended the Reference Committee report to recommend that legislation be "considered" increasing the drinking age to 21.

REPORT OF THE COMMISSION ON MEDICAL SERVICE

The Commission on Medical Service of the South Dakota State Medical Association has held two meetings during the past year, the first on September 17, 1982, and the second on February 26, 1983.

At the first meeting of the Commission, there was a general discussion of the charge of the Commission and its assigned duties as delineated in the Bylaws of the Association. The charge to the Commission as stated in the Bylaws is "The Commission on Medical Service shall have jurisdiction on matters relating to medical education and hospitals; insurance, including programs, prepayment plans, workmen's compensation; rural medical service; traffic safety, and school and public health." Following extensive discussion of the charge of the Commission, it was decided, at this time, to take no positive action in regard to traffic safety or school health. Manpower in the medical profession in South Dakota is reviewed on an ongoing basis, and in the opinion of the Commission, requires no positive action program at this time.

The matter of medical school enrollment was discussed at length. A recommendation was made to the Council that enrollment in the first and second years at the University of South Dakota School of Medicine be limited to no more than fifty

students each year.

It had been suggested to the Commission that sponsorship of a Practice Management Workshop in the state would be beneficial. After review of the costs involved in sponsoring such a workshop presented by the American Medical Association, the rather limited interest expressed, and the unavailability of funds on your
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in most circles, it was the recommendation of the Commission that workshops of this type not be held, but that information be disseminated to interested individuals as to their availability from the American Medical Association in Chicago and at other sites.

The Commission reviewed the Disaster Plan for the State of South Dakota. It is noted that little or no mention is made of medical mobilization in the event of either natural or unnatural disaster. A subcommittee has been appointed to evaluate the Disaster Plan and make recommendations which can be presented to State Civil Defense and disaster planning agencies, with special attention being paid to the integration of available facilities in the state and particularly, the utilization of medical school personnel.

The major focus of the Commission has been that of cost containment. A Subcommittee on Cost Containment was appointed and reported at the last meeting of the Commission. This report has been forwarded to the Council for reaction and guidance. The Commission will continue to study the problems of cost containment, and specific recommendations will be forthcoming.

Respectfully submitted, Loyd R. Wagner, M.D., Chairman Commission on Medical Service

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service and would like to commend the Commission for their work during the year.

REPORT OF THE COMMISSION ON SCIENTIFIC MEDICINE

The Commission on Scientific Medicine did not meet during 1982 and 1983. Inasmuch as it was decided that the annual meeting program should be on risk management, the planning of this program was referred to the Commission on Professional Liability.

Other individual items of business which were referred to the Commission on Scientific Medicine were handled through the mail or directly by the chairman.

Respectfully submitted, A. J. Janusz, M.D., Chairman Commission on Scientific Medicine

The Reference Committee reviewed the report of the Commission on Scientific Medicine. The Reference Committee supports the Committee on Long Range Planning in their survey of the membership regarding future annual meetings and hopes the information received will be of assistance to the Commission on Scientific Medicine in planning future meetings. The Reference Committee recommends acceptance of this report and commends the members of this Commission for their work on behalf of the SDSMA during the past year.

REPORT OF THE COMMISSION ON INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

The Commission met once during the past year on August 25, 1982.

The Commission reviewed two letters received from insurance companies in response to correspondence concerning endorsement of insurance programs by the State Medical Association. The Commission recommended that the executive office check with other insurance companies to determine if another company would be willing to take over the loss of time program currently underwritten by the Harold Diers Company of Omaha, Nebraska. The Commission recommended that no further action to terminate this program be taken.

The Commission recommended that the "Oregon Code of Cooperation" with some minor changes be used as reference material to be provided to the members of the South Dakota State Medical Association.

The Commission reviewed material entitled "The Telephone

in the Doctor's Office," and recommended that this material be included in the reference material to be prepared for members of the South Dakota State Medical Association.

The Commission discussed the Long Range Planning Committee's recommendations that the State Medical Association implement a program to increase and improve the public's image of medicine. The Commission was very willing to assist in the communication and publication of material on this subject.

The Commission reviewed a proposal submitted by IBM for volume discounts on the purchase of typewriters through the Association. The Commission recommended a survey of the membership to see if there was an interest in this type of

program.

During the past year, the following physicians have died: John F. Hill, M.D., Yankton; F. J. Radusch, M.D., formerly of Rapid City; Frederick Rosenfeld, M.D., Hill City; R. E. Lemley, M.D., Rapid City.

The Health Career Grant Fund reported the following activi-

ties during the past twelve months:

32	Alance in Savings Account March 1, 1982		2	4 323 12
	Income		Ψ	7,323.12
	Interest\$	760.05		
		3,763.58		
	Overpayment	26.00		
		4,549.63		4,549.63
			\$	8,872.75
	Expenses			
	Three Grants \$			
	Overpayment	26.00		
	Bank Charge			1,529.00
	\$	1,529.00	\$	7,343.75

lance in Savings Account	
March 1, 1982	\$ 4,323.12
Income	
Interest \$ 760.05	
Principal	
Overpayment 26.00	
\$ 4,549.63	4,549.63
	\$ 8,872.75
Expenses	
Three Grants \$ 1,500.00	
Overpayment	
2.00	1 530 00

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Balance in Savings Account March 1, 1983	\$ 7,343.75
Certificate of Deposit #553 \$14,350.30 #1045 5,629.90	
\$19,980.20 Value March 1, 1983 Interest earned on CD's \$2,127.22	\$19,980.20
Assets \$ 7,343.75 Savings Certificates 19,980.20 Outstanding Loans 7,598.56	\$34,922.51

During the year, the Commission has reviewed each financial report of the State Medical Association, the general account, and the building fund. The Budget and Audit Committee, consisting of the Executive Commission and the Chairman of this Commission, considered and reviewed a budget for the fiscal year 1983-84, and it was submitted to the Council for its consideration and transmitted to the House of Delegates. The proposed budget is attached as part of this report.

Respectfully submitted, Jay W. Hubner, M.D., Chairman Commission on Internal Affairs, Communications & Liaison

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison. The Reference Commission recommends acceptance of this report and commends the members of the Commission for their work on behalf of the SDSMA during the past year.

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PROPOSED BUDGET 1983-84 SOUTH DAKOTA STATE MEDICAL ASSOCIATION

GENERAL	FUND
INCOM	1E

ITEM	BUDGETED 82-83	PROPOSED 83-84
State Dues	\$180,000	\$183,000
Annual Meeting	16,000	25,000
Refunds	5,500	5,500
Car Reimbursement	500	500
Continuing Med. Education	750	750
Salary Reimbursement	12,900	12,900
Other Programs		
7th Dist. Salary Reimb.	1,350	1,350
Equip. Repl. Fund	2,400	2,400
AAFP Salary Reimb.	1,500	1,500
Medical Student Dues	1,000	500
Collection Service	5,000	4,000
Interest	13,000	17,000
Retired Lives Reserve		3,500
	\$239,900	\$257,900

EXPENSES

	BUDGETED	PROPOSED
ITEM	82-83	83-84
Salaries	\$100,000	\$114,000
Social Security	7,000	7,000
Legal & Audit	6,800	7,500
Telephone	4,000	4,000
Office Supplies	8,500	9,000
Ducs & Subscriptions	900	900
Physicians' Travel	12,000	14,000
Annual Meeting	16,000	18,000
Public Relations	6,000	6,500
Journal Subsidy	8,000	6,000
Postage	7,000	7,000
Miscellaneous	100	100
Legislation	6,500	6,000
Staff Travel	11,000	12,000
Insurance	1,500	3,000
Retire/Fringe Benefits	22,000	25,000
Car Operation & Maint.	3,200	3,500
Auxiliary Newsletter	1,300	1,300
Employment Tax	550	800
Continuing Med. Education	750	750
Income Tax	800	800
Replacement of Equipment	1,000	
Med. Student Fund	1,000	500
	\$225,900	\$247,650
Reserve	14,000	10,250
	\$239,900	\$257,900

BUILDING FUND INCOME

ITEM	BUDGETED 82-83	PROPOSED 83-84
Foundation Rent Board of Exam. Rent Interest Income	\$14,040 4,400 10,000	\$14,740 4,400 11,000
Trans. from Gen. Fund	\$28,440	\$30,140

EXPENSES

ITEM	BUDGETED 82-83	PROPOSED 83-84
Salarics Utilitics Taxes & Insurance Maint. & Supplies Legal & Audit	\$15,640 3,300 5,000 3,000 1,500 \$28,440	\$16,140 3,500 5,000 4,000 <u>1,500</u> \$30,140

JOURNAL INCOME

ITEM	BUDGETED 82-83	PROPOSED 83-84
Advertising Subscriptions Refunds Journal Subsidy Miscellaneous	$ \begin{array}{r} \$18,500 \\ 1,000 \\ 720 \\ 8,000 \\ \underline{600} \\ \$28,820 \end{array} $	\$18,500 1,200 720 6,000 <u>600</u> \$27,020
	EXPENSES	
ITEM	BUDGETED 82-83	PROPOSED 83-84
Salaries Legal & Audit Social Security Telephone Postage Office Supp. & Print.	\$ 2,200 100 100 115 2,000 24,285 \$28,820	\$ 2,200 100 100 115 2,000 22,505 \$27,020

REPORT OF THE COMMISSION ON PROFESSIONAL LIABILITY

This past year the Commission met on two occasions in Pierre, South Dakota, July 16, 1982, and October 8, 1982. During those meetings, time was spent reviewing the present status of the legal and political aspects of the Florida statute pertaining to malpractice insurance. The Commission also reviewed multiple approaches toward potential legislation for malpractice and submitted recommendations to the Council for possible legislation to be submitted to the 1983 Legislature.

Information was also reviewed pertaining to the currently active malpractice cases within the state of South Dakota. The chairman of the Commission and representation from the state executive office attended a Risk Management Seminar in North Dakota early in the year and reported on the favorable results of that seminar.

The Commission proceeded to make plans and recommendations to the Commission on Scientific Medicine regarding the advisability of planning a Risk Management Seminar, to be held in conjunction with the annual meeting of the State Medical Association in 1983. Extensive time was devoted to the planning of this meeting during the October 8 session. We hope this type of risk seminar may be of value to all specialties within the field of medicine in the state of South Dakota and be of benefit to all practicing physicians.

Respectfully submitted, Morris Radack, M.D., Chairman Commission on Professional Liability

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends acceptance of this report and commends the members of the Commission for their work on behalf of the SDSMA during the past year.

REPORT OF THE COMMITTEE FOR CONTINUING MEDICAL EDUCATION

No meetings were held this past year and no new sites have been approved for continuing medical education. The Watertown and Rapid City programs continue to be active and busy and will be reviewed for renewal this year.

Respectfully submitted, K. Gene Koob, M.D., Chairman Committee for Continuing Medical Education

The Reference Committee reviewed the report of the Committee for Continuing Medical Education and recommends acceptance of this report.

REPORT OF THE GRIEVANCE COMMISSION

The Grievance Commission has acted upon a total of 16 individual grievances during the year. Ten of the grievances were found to be unwarranted and the physician's care was deemed correct in each of these instances. The remainder of the cases involved a variety of complaints, some of which were instituted by patients against physicians who failed to transfer adequate records.

In regard to transfer of records, the Commission has looked into this both medically and legally, and would again advise the membership that when a patient asks for transfer of records, the records themselves must be totally transferred if that is the wish of the patient. It is perfectly all right to transfer a narrative summary, unless the patient would designate that photocopies of the records be transferred.

In last year's report from the Grievance Commission, I noted that a high incidence of the grievances came about because of criticism of one doctor by another, many times without full knowledge of what the previous treatment had been. This problem continues to exist. It should be noted that a high incidence of the grievances have come from one specific area in South Dakota, and, of these grievances, many have been instituted because of questionable criticisms of a fellow physician. It should also be of interest to the membership that there is a higher incidence of malpractice suits being instituted in this same area.

The Commission wants to make very clear that we do not believe poor medical procedures should be covered up in any way, and we believe that doctors should continue to monitor their peers whenever possible. However, it is imperative that when this criticism is made, it is well documented and that all of the facts are known.

As a last thought, the Commission would suggest that all of us should practice carefully, with great sympathy for the patient, and keep good records.

Respectfully submitted, Duane B. Reaney, M.D., Chairman Grievance Commission

The Reference Committee reviewed the report of the Grievance Commission and recommends acceptance of the report reiterating that when a physician criticizes a peer, "it is important that when this criticism is made, it is well documented and that all facts are known."

REPORT OF THE LONG RANGE PLANNING COMMITTEE

The Long Range Planning Committee had one meeting this past year. We discussed promotion of political awareness among physicians and their spouses with recommendations being made. We began working on a questionnaire to survey physician's concepts of medical needs of the people in addition to the physician's needs. I am in hopes that the information gained from the survey will give us some ideas to follow up regarding our long-range planning for the South Dakota Medical Association.

Respectfully submitted, G. D. Loos, M.D., Chairman Long Range Planning Committee

The Reference Committee reviewed the report of the Long Range Planning Committee and recommends acceptance of the report.

REPORT OF THE ARCHIVES AND HISTORY COMMISSION

The Commission on Archives and History had no formal meeting during the year but material for the archives are being gathered and filed at our executive office.

Respectfully submitted, C. J. McDonald, M.D., Chairman Archives and History Commission

The Reference Committee reviewed the report of the Archives and History Commission and recommends acceptance of the report.

REPORT OF THE LIAISON COMMITTEE

The Liaison Committee met twice during the past year and will meet again in early May. These meetings with representatives of the Health Department are called to discuss matters of mutual concern between the Health Department and practicing physicians in South Dakota. The sessions have been quite productive and helpful in resolving potential problems.

Members of this committee are to be commended for their thoughtful consideration and for the time expended on behalf of the Association. Reports of the Liaison Committee meetings have been submitted and discussed with the Council on a regular basis

Respectfully submitted, John T. Elston, M.D., Chairman Liaison Committee

The Reference Committee reviewed the report of the Liaison Committee and recommends acceptance of the report.

REPORT OF THE SOUTH DAKOTA POLITICAL ACTION COMMITTEE

1982 was a successful year for SoDaPAC in terms of supporting successful candidates for the South Dakota legislature. In all, SoDaPAC supported 66 candidates of which 56 were successful — 85%. Contributions to the candidates ranged from \$50 to \$250.

SoDaPAC also supported Governor Janklow's re-election campaign and requested maximum AMPAC support for Clint Roberts' re-election bid

Roberts' re-election bid.

Your Board of Directors is now looking to the election of 1984 as a crucial year for medicine in continuing its strong voice in South Dakota politics. The Board has several objectives to accomplish, including: renewal of as many SoDaPAC past members as possible; encouraging more members to become sustainers; recruiting new members; and electing worthy candidates who understand and appreciate the unique contributions made by medicine.

Doctors, we need you and your spouses' help to achieve these objectives. In 1982, only one of every five State Medical Association members belonged to SoDaPAC! We can and must do better.

Twenty years ago, in 1963, the South Dakota Physicians Committee became the South Dakota Political Action Committee . . . medicine stands to lose so much if we as physicians collectively do not become involved in making the next twenty years as good as the past twenty years. Join us.

Respectfully submitted, T. J. Wrage, Jr., M.D. Chairman, SoDaPAC

The Reference Committee reviewed the report of the South Dakota Political Action Committee and recommends acceptance of the report.

REPORT OF THE BOARD OF DIRECTORS OF THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT ASSOCIATION

The Board of Directors of the South Dakota Medical School Endowment Association met as prescribed by the Association Bylaws during the Annual Meeting of the South Dakota State Medical Association, which was held in Rapid City on May 20-23, 1982. Throughout the year business has been conducted by telephone or mail through the coordination of the staff of the Medical Association.

The goals of the Endowment Association continue to be the support of deserving medical students of the University of South Dakota School of Medicine through loans made at reasonable rates of interest and support of the office of the Dean of the school in essential activities for which there are not other funds. Assets of the Association will support loans approximating a total of \$20,000 per year. The loss of State and Federal funds during the past two years has placed a greater demand on the

Endowment Association. For part of the current academic year no public funds were available to students. Emergency advances were required from the Endowment Association to keep some in school. These advanced funds were repaid when other funds became available later in the year.

Contributions to the Endowment Fund are encouraged throughout the year. A solicitation is conducted in the Autumn. The practicing physicians of South Dakota continue to be the major contributors. The Endowment Association remains an essential function of the South Dakota State Medical Associa-

Respectfully submitted, The Board of Directors: Joseph N. Hamm, M.D., Chairman Bruce H. Allen, M.D. Robert R. Giebink, M.D. Warren L. Jones, M.D. Bruce C. Lushbough, M.D. Theodore H. Sattler, M.D. Gerald E. Tracy, M.D.

The Reference Committee reviewed the report of the Board of Directors of the South Dakota Medical School Endowment Association and recommends acceptance of the report.

REPORT OF THE PRESIDENT OF THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT ASSOCIATION

The President has attended the regular and ad-hoc meetings of the South Dakota Medical School Endowment Association throughout the year. He wishes to thank the Board of Directors and the physicians of South Dakota who have given support to medical education through the Endowment Association during the past year.

> Respectfully submitted, Joseph N. Hamm, M.D., President South Dakota Medical School **Endowment Association**

The Reference Committee reviewed the report of the President of the South Dakota Medical School Endowment Association and recommends acceptance of the report.

MINUTES OF SOUTH DAKOTA MEDICAL SERVICE, INC. CORPORATE BODY MEETING

Ramada Inn Sioux Falls, South Dakota June 3, 1983, 9:15 a.m.

Chairman Ortmeier called the meeting of the Corporate Body of South Dakota Medical Service, Inc., to order at 9:15 a.m. on June 3, 1983, at the Ramada Inn in Sioux Falls, South Dakota.

Upon roll call, the following members of the Corporate Body of the South Dakota Medical Service, Inc., were present:

Drs. Durward Lang, J. N. Hamm, Howard Saylor, Jr., W. O. Rossing, A. J. Barrett, G. E. Tracy, Russell Harris, B. C. Lushbough, A. A. Lampert, Jr., David Buchanan, R. G. Gere, Guy Tam, Larry Sittner, Lowell Hyland, Michael Pekas, Richard Porter, Frank Messner, Roger Millea, Robert L. Ferrell, James Jackson, M. G. Thompson, Charles Pelton, James Larson, Parry Nelson, Charles S. Roberts, Richard Holm, John Davis, William G. M. Huet, Ravi Kapur, Walter Baas, T. J. Bhatti, V. Brandenburg, George Bruins, Michael Ferrell, David Ohrt, Ronald Wyatt, Gail Benson, Jeffrey Hagen, Jerry Freeman, Roger Stoltz, Jay Hubner, R. J. Foley, Michael McVay, Richard P. Renka, James Kullbom, N. R. Whitney, Robert Stiehl and David Yecha.

Dr. Harris moved that the roll call taken by the Secretary of the Medical Association be accepted by the Corporate Body. The motion was seconded by Dr. Lang. Upon voice vote, the same was approved unanimously.

A quorum being present, the Chairman declared the annual meeting of the membership of the Corporate Body of South Dakota Medical Service, Inc., to be duly in session for the transaction of business.

Dr. Lang moved that reading of the minutes of the last meeting of the Corporate Body, being the 1982 Annual Meeting, be waived, the same having been published in the Handbook and previously mailed to each member. Such motion was seconded by Dr. Lushbough. Upon voice vote, the same was approved unanimously.

Dennis Ortmeier introduced the board members who were present to the Body. He also introduced staff members and the medical director.

Chairman Ortmeier presented the Chairman's message to the Corporate Body as contained in writing in the Delegates' Handbook. He noted the financial difficulties South Dakota Blue Shield was encountering several years ago and the improvements that have recently been made therein through cost containment programs, more aggressive sales, and Commissioner approved premium increases. He especially cited Medigap and Medigap Plus and new groups such as the South Dakota Farmers Union. He thanked the physicians of South Dakota for their cooperation and gave special recognition to Dr. Paul Aspaas for twelve years of service to the South Dakota Blue Shield Board as well as to John Olson who resigned from the Board after serving ten years. He said the Corporation is the richer for their contributions.

Dr. Tracy moved the acceptance of the Chairman's Report. The motion was seconded by Dr. Saylor. Upon voice vote, the same was approved unanimously.

Chairman Ortmeier called for consideration of the next agenda item, entitled "Financial Report" and called upon Ben Johnson to present the same. Mr. Johnson referred the corporate body to the 1982 Annual Report contained in their Delegate's Handbook. First he pointed out that the total assets of the corporation on January 1, 1983, were \$6,489,977 as compared to the prior years \$5,797,000. The Blue Shield had on investment approximately \$5.5 million which is where the substantial investment income reported came from. The unassigned surplus at the end of 1982 was \$1,819,000. He noted that total income and payout on claims showed that 83.3% of all Blue Shield dollars went out to pay patient claims. Claims paid as compared to premium income was 86.3%.

Ben Johnson asked if anybody in the Body had any questions on the financial report. Dr. Hamm asked where the two year comparative sheet was. Ben stated that the comparative figures were not contained in the Financial Report they had but that he would provide any figures from past reports which were desired. He noticed that in 1981 Blue Shield had a loss of \$511,000 as compared to a gain in 1982 of \$456,000.

Dr. Lushbough moved approval of the Financial Report. The motion was seconded by Dr. Harris. Upon voice vote, the same

was approved unanimously.

The Chairman called for the report of the Nominating Committee. The Chairman of the Nominating Committee, Dr. Dean, reported that the Committee consisting of himself, Drs. Hagen, Seaman, Gere and Art Lampert, Jr., submitted the following persons' names:

1. Nominated for reelection to the Blue Shield Board of Direc-

Ralph Nauman of Gettysburg, John Stransky, M.D., of Watertown,

for three-year terms.

Nominated for original election to the Blue Shield Board of Directors:

Winston Odland, M.D., of Aberdeen,

Glen Waltner of Freeman, Chairman of the First National Bank of Freeman,

each for a three-year term to replace Dr. Aspaas, who is not eligible for reelection, and John Olson, who resigned from the Board when he left the position as President of South Dakota Blue Shield.

The foregoing report was submitted by Drs. Hagen, Seaman, Gere and Art Lampert, Jr., and Roscoe Dean as Chairman.

Dr. Lang moved that the nominations cease and the Secretary be instructed to cast a unanimous ballot for the nominees as

directors of the corporation. The motion was seconded by Dr. Larson. Upon voice vote, the same was approved unanimously.

The Chairman called upon Ben Johnson to make awards authorized by the Directors. He requested Dr. Aspaas to come forward.

Dr. Aspaas was presented an award in the form of a plaque noting his twelve years of service to South Dakota Blue Shield. Dr. Aspaas stated that being on the Blue Shield Board was a great learning process and he was impressed with its leadership.

Mr. Johnson requested that John Olson come forward. Mr. Olson served as president of Blue Shield during 1982 and also for ten years on the Board of Directors. Mr. Olson also received

a plaque in recognition of his service.

Mr. Olson expressed his thanks for the cooperation he received as a board member and the year he served as president. He stated that the support from the medical community was an underlying foundation which helped Blue Shield to survive a number of problems during the years he served as a director of Blue Shield. He thanked the Body for their help and consideration.

Chairman Ortmeier asked if any member of the body cared to present any other old business. There being no old business presented, he asked if any member of the body had any questions for members of the staff or any of the Blue Shield directors. No questions were asked from the floor.

The Chairman requested Pete Galindo to explain to the group what was happening on the new physician participation agreements. Mr. Galindo noted that previously letters that had gone out voiding the prior physician participation agreements as of July 1, 1983, and asking physicians to sign new agreements. He noted that currently Blue Shield has received approximately 320 of the new agreements with physicians and it appears that there will be 400 by July 1. That would be approximately 65% of the physicians in private medical practice in South Dakota.

He asked if any member of the Body had any questions from the floor. Dr. Ferrell questioned the figures on physicians who had signed the new agreements. He stated he found the agree-

ment very distasteful.

Dr. Ferrell moved that the provision in the Physician Participation Agreements stating that a participating physician would accept the amount paid by Blue Shield as payment in full for services to covered patients be deleted from all present and future Physician Participation Agreements. The motion was seconded by Dr. Harris. A substantial discussion on the motion followed, after which Dr. Tracy moved the question. Dr. Barrett moved that the voting be by secret ballot. Such motion was seconded by Dr. Tracy. Upon voice vote taken, the Chairman ruled that the secret ballot motion prevailed and the vote would therefore be by secret ballot.

Ballots were passed to all members of the Corporate Body and each was instructed to mark their ballot for or against the motion of Dr. Ferrell. The Chairman explained that a "Yes" vote was in favor of the motion to delete the provision from the Physician Participation Agreement requiring a participating physician to accept Blue Shield payment as payment in full and a "No" vote

was a vote to retain such provision.

At the conclusion of voting, Dr. Lushbough and Dr. Renka were asked by the Chairman to serve as ballot counters.

Dr. Lushbough and Dr. Renka counted the ballots and tallied the same. They informed the Chairman that a majority of the votes cast were "No" ballots and therefore against the motion.

The Chairman announced that the motion failed.

Dr. Harris requested that Blue Shield review terminology in certificate holder contracts and the forms used by Blue Shield. As an example, he cited the notation sent to patients on some physician charges that such charges were "more than the allowable charge." He stated that such terminology created substantial problems. He requested that consideration be given to changing the wording to "more than the allowable reimbursement." He cited this as an example of terminology changes that could eliminate problems. After discussion, Dr. Harris moved that a committee consisting of members from both the Blue Shield Board and the South Dakota State Medical Council, be formed to study wording in Blue Shield documents to see if

improvement in language could eliminate certain problems to the benefit of all parties, including the patient. The motion was seconded by Dr. Lushbough. Upon voice vote, the same was approved unanimously.

Chairman Ortmeier asked if there was any other business to come before the Corporate Body. No other business was presented. Dr. Saylor moved that the meeting be adjourned. The motion was seconded by Dr. Harris. Upon voice vote, the same was approved unanimously.

The meeting was adjourned at 9:45 a.m.

John H. Zimmer Secretary

PRESIDENTIAL OATH OF OFFICE

I SOLEMNLY SWEAR THAT I shall carry out the duties of the President of the South Dakota State Medical Association to the best of my ability. I shall strive constantly to maintain the ethics of the medical profession and to promote the public health and welfare. I shall dedicate myself and my office to improving health standards and to the task of bringing increasingly improved medical care to the people of South Dakota. I shall uphold the Constitution and Bylaws of the AMA and the South Dakota State Medical Association. I shall champion the cause of freedom in medical practice and freedom for all my fellow Americans.

I do solemnly swear that I will discharge the duties of this office to the best of my ability, so help me God.

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Mary Helen Pelton, Ph.D. The Office of Rural Health University of North Dakota School of Medicine Grand Forks, ND 58201 Phone: (701) 777-3848

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DISTINGUISHED SERVICE AWARD

Started in 1951 — T. F. Riggs, M.D., Pierre (deceased)

1952 — H. Russell Brown, M.D., Watertown (deceased)

1953 — Guy Van Demark, M.D., Sioux Falls (deceased)

1954 — J. C. Ohlmacher, M.D., Vermillion (deceased)

1955 — R. G. Mayer, M.D., Aberdeen (deceased)

1956 — J. C. Ohlmacher, M.D., Vermillion (deceased)

1957 — W. E. Donahoe, M.D., Sioux Falls (deceased)

1958 — Drs. J. C. Hagin (deceased), M. W. Pangburn (deceased), and James DeGeest, Miller

1958 — J. F. Brenckle, M.D., Superior, Wisc. (deceased)

1958 — Mrs. Agnes Holdridge, Madison

1959 — Walter L. Hard, Ph.D., Vermillion

1959 — Rev. and Mrs. Robert O. Bates, Sturgis 1959 — R. M. Kilgard, M.D., Watertown (deceased)

1960 — L. J. Pankow, M.D., Sioux Falls (deceased)

1961 — Gregg M. Evans, Ph.D., Custer

1962 — Edward Shaw, Ph.D., Vermillion (deceased)

1963 — Arthur A. Lampert, M.D., Rapid City

1964 — John C. Foster, Phoenix, Arizona

1965 — A. P. Reding, M.D., Marion

1966 — Mrs. C. Rodney Stoltz, Watertown

1967 — Mrs. William Fish, Watertown

1968 — G. J. Bloemendaal, M.D., Ipswich

1969 — F. W. Haas, M.D., Yankton (deceased)

1970 — Paul Bunker, M.D., Aberdeen (deceased)

1971 — E. T. Lietzke, M.D., Beresford (deceased)

1972 — C. B. McVay, M.D., Yankton

1973 — G. E. Tracy, M.D., Watertown

1974 — J. A. Muggly, M.D., Madison (deceased)

1975 — Harvey Wollman, Hitchcock

1976 — R. H. Quinn, M.D., Sioux Falls

1977 — E. H. Heinrichs, M.D., Vermillion

1978 — John Olson, Sioux Falls, and Evans Nord, Sioux Falls

1979 — Helen Jane Hare, M.D., Rapid City

1980 — Warren Jones, M.D., Sioux Falls 1981 — Saul Friefeld, M.D., Brookings

1982 — G. Robert Bartron, M.D., Watertown

1983 — Oscar J. Mabee, M.D., Mitchell

COMMUNITY SERVICE AWARD

1961 — R. A. Buchanan, M.D., Huron (deceased)

1962 — Roland F. Hubner, M.D., Yankton

1963 — George W. Mills, M.D., Wall (deceased)

1964 — John C. Hagin, M.D., Miller (deceased)

1965 — Alonzo P. Peeke, M.D., Volga

1966 — Hugo C. Andre, M.D., Vermillion (deceased)

1967 — G. Robert Bartron, M.D., Watertown

1968 — M. M. Morrissey, M.D., Pierre (deceased)

1969 — N. J. Sundet, M.D., Kadoka (deceased) 1970 — W. H. Saxton, M.D., Huron (deceased)

1971 — R. E. Van Demark, M.D., Sioux Falls

1972 — R. H. Hayes, M.D., Wall

1973 — B. F. King, M.D., Aberdeen (deceased)

1974 — M. C. Tank, M.D., Brookings

1975 — Karl Wegner, M.D., Sioux Falls 1976 — John T. Elston, M.D., Rapid City

1977 — W. F. Stanage, M.D., Pierre

1978 — C. S. Roberts, Jr., M.D., Brookings 1979 — C. J. McDonald, M.D., Sioux Falls

1980 — E. A. Johnson, M.D., Milbank

1981 — J. A. Muggly, M.D., Madison (deceased)

1982 — Robert R. Giebink, M.D., Sioux Falls

1983 — Theodore H. Sattler, M.D., Yankton

AESCULAPIUS AWARD

1966 — Paul R. Leon, M.D. Walter Miller, M.D., Aberdeen

1968 — H. Phil Gross, M.D., Sioux Falls

FIFTY YEAR CLUB MEMBERS

C. V. Auld, Plankinton (deceased)

G. J. Bloemendaal, M.D., Ipswich

W. C. Brinkman, M.D., Sisseton (deceased)

R. A. Buchanan, M.D., Huron (deceased) John L. Calene, M.D., California (deceased)

Myrtle Carney, M.D., Ft. Worth, Texas

J. C. Clark, M.D., Sioux Falls (deceased)

F. L. Class, M.D., Huron (deceased)

M. E. Cogswell, M.D., Wolsey (deceased)

J. Cook, M.D., Bonesteel (deceased) G. I. W. Cottam, M.D., Sioux Falls

Harold L. Crane, M.D., Avon, Conn. (deceased)

S. A. Donahoe, M.D., Sioux Falls (deceased)

W. E. Donahoe, M.D., Sioux Falls (deceased)

J. A. Eckrich, Sr., M.D., Aberdeen

V. W. Embree, M.D., Pierre (deceased) W. D. Farrell, M.D., Aberdeen (deceased)

R. B. Fleeger, M.D., Lead (deceased)

R. R. Fisk, M.D., Flandreau (deceased)

F. W. Freyberg, M.D., Mitchell (deceased)

E. E. Gage, M.D., Sioux Falls (deceased) D. A. Gregory, M.D., Glasgow, Mont.

E. H. Grove, M.D., Arlington (deceased)

J. C. Hagin, M.D., Miller (deceased)

Lyle Hare, M.D., Spearfish (deceased) John F. Hill, M.D., Yankton (deceased)

J. A. Hohf, M.D., Yankton (deceased)

F. S. Howe, M.D., Deadwood (deceased)

A. H. Hovne, M.D., Salem (deceased)

A. S. Jackson, M.D., Rapid City (deceased)

R. J. Jackson, M.D., Hot Springs (deceased)

J. A. Jacotel, M.D., Milbank (deceased)

G. T. Jordan, M.D., Vermillion (deceased)

F. F. Keene, M.D., Wessington Springs (deceased)

Ray Lemley, M.D., Rapid City (deceased)

J. H. Lloyd, M.D., Mitchell

O. J. Mabee, M.D., Mitchell

P. V. McCarthy, M.D., Aberdeen (deceased)

G. W. Mills, M.D., Wall (deceased)

B. C. Murdy, M.D., Aberdeen (deceased)

T. F. O'Toole, M.D., Rapid City (deceased)

N. T. Owen, M.D., Rapid City (deceased)

L. L. Parke, M.D., Canton (deceased)

A. P. Peeke, M.D., Volga

M. O. Pemberton, M.D., Deadwood (deceased)

R. J. Quinn, M.D., Sioux Falls (deceased)

F. J. Radusch, M.D., California (deceased)

T. B. Ranney, M.D., Aberdeen (deceased)

T. F. Riggs, M.D., Pierre (deceased)

I. R. Salladay, M.D., Ft. Meade (deceased)

W. H. Saxton, M.D., Huron (deceased)

H. L. Saylor, M.D., Huron (deceased)

C. E. Sherwood, M.D., Brookings (deceased)

Arthur W. Spiry, M.D., Mobridge

Myron Tank, M.D., Brookings

F. J. Tobin, M.D., Mitchell (deceased)

Leonard W. Tobin, M.D., Mitchell

J. S. Tschetter, M.D., Huron (deceased)

Paul Tschetter, M.D., Huron

F. W. Valkenaar, M.D., Chancellor (deceased)

G. E. Van DeMark, M.D., Sioux Falls (deceased)

H. P. Volin, M.D., Lennox (deceased)

C. H. Weishaar, M.D., Aberdeen (deceased)

J. R. Westaby, M.D., Madison (deceased)

G. E. Zimmerman, M.D., Missoula, Montana (deceased)

C. B. ALFORD AWARD

1974 — Roscoe Dean, M.D., Wessington Springs

1975 — Gerald Tracy, M.D., Watertown

1976 — Robert Westaby, M.D., Hot Springs

1977 — Robert VanDemark, M.D., Sioux Falls

1978 — Howard Saylor, M.D., Huron

1979 — J. D. Bailey, M.D., Rapid City

1980 — John T. Elston, M.D., Rapid City

1981 — T. H. Sattler, M.D., Yankton

1982 — Bedford T. Otey, M.D., Flandreau

1983 — Robert H. Quinn, M.D., Sioux Falls

SDSMA Council Meetings

Friday, September 23 Howard Johnson Motor Lodge Sioux Falls, SD 9:00 a.m.-5:00 p.m.

Friday, November 18 Holiday Inn Centre Sioux Falls, SD 9:00 a.m.-5:00 p.m.

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S President's Page

Several organizations solicit donations for medical education in South Dakota. Their names and objectives are so similar as to cause confusion. At the annual meeting it was suggested that a brief review of the fund raising institutions, their sponsorship and objectives would be helpful. This précis will hopefully suffice.

The South Dakota Medical School Endowment Association was first to be established in 1949. It continues to function as a corporation under the aegis of the South Dakota Medical Association, to provide loans to students at low rates of interest, currently six percent. Presently outstanding loans total more than \$269,000. By far the most donations are made by physicians practicing in South Dakota. In the last year 139 physicians gave nearly \$20,000 to the endowment. Loans are payable one year after completion of postgraduate training at which time interest increases to one percent above prime rate. The Endowment Association has provided "levering" money to acquire federal loans for students, emergency expenses for students, and the only unrestricted funds for the office of the Dean of the Medical School. Early in 1983 a loan was made through medical school faculty to pay student expenses when no other funding was available. This loan has been repaid.

In 1950 the American Medical Association established the Education and Research Fund (AMA-ERF). Physicians, auxiliary members and others have been solicited through state medical societies. Donors are allowed to designate the school which will receive their gift. Money has been used to guarantee loans made to medical students by banks, and for the unrestricted use of the designated medical school. The AMA-ERF has distributed more than \$36 million during the past 32 years. In the late 1970's high interest rates made bank loans unavailable. AMA-ERF funds collected are retained to guarantee outstanding loans. A new AMA-ERF has now been established. Gifts will be allocated as in the past. Schools will get regular reports and a gift



once each year. Money will support student aid programs and loan funds. No expenses will be deducted from the gift.

The University of South Dakota School of Medicine Alumni Foundation was launched in 1980 by funds supplied by the South Dakota Medical School Endowment Association. Expenses were subsidized for three years. The Alumni Foundation solicits from graduates of the Medical School living within and outside of the state. It hopes to build to a fund of \$250,000 by the end of 1984. Generated income and donations will then be distributed to the USD School of Medicine.

The University of South Dakota Foundation emerged in 1974 through the union of two alumni organizations — the first established in 1920. The Foundation promotes fund raising and is eligible to accept gifts for betterment of the University. All graduates are solicited. The Century 2 Campaign currently underway is to terminate on June 30, 1983.

In 1983 our School of Medicine received \$21,157 from the AMA-ERF, largely from the labor of our Auxiliary. Only twelve schools in America got more. Wonderful South Dakota is not "last in everything!"

Joseph N. Hamm, M.D., President South Dakota State Medical Association

South Dakota State Medical Association Roster — 1983 Membership by Districts

ABERDEEN DISTRICT No. 1

DISTRICT No. 1			
Pres., Carlton Kom, M.D. Sec., Robert Brown, M.D.			
Albano, Paterno C. Aberdeen Alexander, James R. Aberdeen Altman, Stanley B. Aberdeen Andersen, Calvin F. Aberdeen Anderson, Esther E. Aberdeen Bachmayer, Jay D. Aberdeen Bartholomew, Kenneth A. Faulkton Berg, Sterling Redfield *Bloemendaal, Gerrit J. Ipswich Brevik, Alan K. Redfield Broadhurst, Kennon E. Aberdeen Brown, Robert H. Aberdeen Bunker, Thomas G. Aberdeen Carter, Peter B. Aberdeen Chang, Joe P. Aberdeen Chavier, Juan R. Aberdeen Chen, Cheng-Fu Aberdeen Christopher, John R. Aberdeen Christopher, John R. Aberdeen Eckrich, Jerome A., Jr. Aberdeen Eckrich, Jerome A., Sr. Aberdeen	Fahrenwald, Myron E., Aberdeen Gerber, Bernard C. Aberdeen Harlow, Mark C. Aberdeen Hart, Harvey J. Aberdeen Heinemann, Phyllis E. Aberdeen Hovland, James I. Aberdeen Huber, Joel B. Redfield Jahoda, Diane Douglas, GA Janusz, Albin J. Aberdeen Kazi, K. Stephen Aberdeen Kom, Carlton J. Aberdeen Kosse, Karl H. Aberdeen Leon, Paul R. Aberdeen McFee, John L. Ipswich McGee, Robert C. Aberdeen McIntosh, George F. Eureka Myrmoe, Arlin M. Aberdeen *Norgello, Vikentijs Odland, Winston B. Aberdeen Ostrowski, Susan M. Eureka Ottenbacher, John C. Bowdle	Patterson, David M. Redfield Pelton, Charles L. Aberdeen Redmond, Warren J. Aberdeen Rodine, John C. Aberdeen *Rudolph, E. A. California Sanders, Mary E. Aberdeen Scheffel, Alvin R. Redfield Seaman, David Aberdeen Shaw, Wayne R. Aberdeen Shinghal, Kumud K. Aberdeen Shinghal, Pramod Aberdeen Shousha, Alfred Britton Steele, Granville H. Aberdeen Stopple, John A. Aberdeen Soweeny, William T. Aberdeen Tan, Raymundo T. Aberdeen Tan, Raymundo T. Aberdeen Unite, Ismael H. Aberdeen Unite, Ismael H. Aberdeen Welge, Barry G. Aberdeen Zvejnieks, Karlis Aberdeen Zvejnieks, Karlis	
	WATERTOWN DISTRICT No. 2		
Pres., Richard McClaflin, M.D.	Vice Pres., James Horning, M.D.	Sec., G. E. Tracy, M.D.	
Allen, Stanley W., Jr. Watertown Argabrite, John W. Watertown Bartron, G. Robert Watertown *Bartron, Harry J., Jr. Watertown Brakss, Valdis Watertown Brown, Bradley A. Watertown Clark, Carroll J. Watertown Desai, Bhasker J. Watertown Engelhart, Kenneth E. Watertown Fedt, Donald N. Watertown Frazer, Paul D. Clear Lake Guddal, W. Nicol Watertown	Hanson, Bernie H. P. Watertown Horning, James R. Watertown Hughes, Howard D. Clear Lake *Huppler, E. G. Minnesota Larson, James C. Watertown Larson, Paul M. Watertown Likness, Clark W. Watertown McClaflin, Richard R. Watertown Meyer, Robert J. Watertown Michieli, Jose C. Watertown Nelson, Parry S. Watertown	Peterson, Linda H. Watertown Piro, David F. Watertown Rittmann, John E. Watertown *Rousseau, Maurice C. Watertown Ruggles, James G. Watertown *Stoltz, C. Rodney Sioux Falls Stransky, John J. Watertown Thompson, Marion C. Watertown Tracy, Gerald E. Watertown Ulrickson, Mary L. Watertown Wrage, Theodore J., Jr. Watertown	
	MADISON-BROOKINGS DISTRICT No. 3		
Pres., Samuel Bandiera	Vice Pres., Kim Wilde, M.D. Sec	., Richard Holm, M.D.	
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Javurek, Anthony J Sioux Falls	Nelson, Robert E Sioux Falls	Solberg, Lloyd E Sioux Falls
Johnson, Beth L Sioux Falls	Nice, Richard F Sioux Falls	Soye, Andrew I Sioux Falls
Johnson, Dennis L Sioux Falls	Nielsen, James L Dell Rapids	*Stahmann, Fred S Sioux Falls
Johnson, Robert C Sioux Falls	Nordstrom, Donald G Sioux Falls	Stassen, Michael D Sioux Falls
Jones, Warren L Sioux Falls	Oakland, James A Sioux Falls	*Steiner, Peter K California
Justice, Michael W Dell Rapids	O'Brien, Charles P Sioux Falls	Stensland, Vernon H Sioux Falls
Kalda, Ellison F., II Sioux Falls	O'Brien, Peter J Sioux Falls	*Stern, Charles A California
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Knowles, Roy C Sioux Falls	Orr, Russell T Sioux Falls	Tschetter, Loren K Sioux Falls
Knudson, Donald H Sioux Falls	Ortmeier, Denny G Sioux Falls	Tschetter, Richard T Sioux Falls
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Lakstigala, Peters E Sioux Falls	Payne, Harlan A Sioux Falls	VanderWoude, Larry B. Sioux Falls
		Villa, Jose P Freeman
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Lang, Terry A Sioux Falls	Petereit, Martin F Sioux Falls	Vogt, H. Bruce Sioux Falls
Langdon, John G Sioux Falls	Peters, Edward H Sioux Falls	Volin, Verlynne V Sioux Falls
Lankhorst, Barry J Sioux Falls	Peterson, Karl G Sioux Falls	Wagner, Loyd R Sioux Falls
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Larke, Daryl S Sioux Falls	Petres, Anthony Salem	Waltner, Lonnie L Bridgewater
Larson, Leland J Sioux Falls	Pitt-Hart, Barry T Sioux Falls	Walton, Jerry L Sioux Falls
*Leander, Richard B Sioux Falls	Putnam, Wesley D Sioux Falls	Watson, William V Sioux Falls
Lee, Si Gaph Sioux Falls	Quale, James L Sioux Falls	Wegner, Karl H Sioux Falls
Looby, Thomas L Sioux Falls	Quinn, Robert H Sioux Falls	White, Thomas C Sioux Falls
Loos, Gerald D Sioux Falls	Randall, Bradley B Sioux Falls	Wierda, Daryl R Sioux Falls
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Madison, Dean L Sioux Falls	Reynolds, James R Sioux Falls	Woods, Gail L Sioux Falls
Magnuson, Gregory L Sioux Falls	Richards, George A Sioux Falls	Wyatt, George W Sioux Falls
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Bean, David W Yankton	Jameson, G. Malcolm Yankton	Price, Ronald D Armour
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Foley, Robert J Tyndall		
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Gunderson, Dale E Yankton	*McVay, Chester B Yankton McVay, Michael R Yankton	Reding, Arthur P Marion Rhoades, Marques E Yankton

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McVay, Michael R. Yankton

Messner, Frank D. Yankton
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Neumayr, Robert J. - Yankton

Nutt, Roger W. Yankton Olson, Thomas H. Vermillion

Pascale, Carl C. Centerville
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Rhoades, Marques E. Yankton

*Rioades, Marques E. Tankton

*Riesberg, Elsa Aberdeen
Saloum, Herbert T. Tyndall
Saoi, Nicasio B. Yankton
Sattler, Theodore H. Yankton

*Sebring, Floyd U. Vermillion

Smith David A. Vankton

Smith, David A. Yankton Stanage, Willis F. Pierre

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Gunderson, Dale E. Yankton
Halverson, Kenneth Yankton
Heinrichs, Eberhard H. Vermillion
Holzwarth, David R. Yankton
Harle Birberd W. Wagner

Honke, Richard W. Wagner
Honke, Richard W., II. Wagner
Hubner, Jay W. Yankton
*Hubner, Roland F. Yankton

Stephenson, Daryl R. Yankton Sternquist, John C. Yankton Stevens, Julie C. Yankton	Tidd, John T. Yankton Tuan, Chung H. Yankton	Willcockson, John R Yankton Willcockson, Thomas H Yankton
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Ahrlin, Hollis L	Rapid City
Ahrlin, H. Lee, Jr. Ahrlin, Hollis L. Albano, Paterno C.	. Aberdeen
*Alcorn, Floyd	Sioux Falls
*Alcorn, Floyd	. Aberdeen
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Allen, Bruce H. Allen, Robert G. Allen, Stanley W., Jr.	Watertown
Altman, Stanley B	. Aberdeen
Altman, Stanley B Alvine, Frank G	Sioux Falls
Amundson, Loren H	Sioux Falls
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Bandiera, Samuel J Bareis, Reuben J Barker, John D., Jr. Barlow, John F Barnett, George L	Brookings Rapid City Sioux Falls Sioux Falls Sioux Falls
Bandiera, Samuel J. Bareis, Reuben J. Barker, John D., Jr. Barlow, John F. Barnett, George L. Barrett, Arthur J.	Brookings Rapid City Sioux Falls Sioux Falls Sioux Falls Rapid City
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Bandiera, Samuel J. Bareis, Reuben J. Barker, John D., Jr. Barlow, John F. Barnett, George L. Barrett, Arthur J. Bartholomew, Kenneth A. Bartron, G. Robert *Bartron, Harry J., Jr. Bauman, Randell, E. Bean, David W.	Rapid City Sioux Falls Sioux Falls Sioux Falls Sioux Falls Rapid City Faulkton Watertown Watertown Rapid City Yankton
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Bell G Robert	DeSmet
Relyea Mark E	Huron
Penson Coil M	Ciarry Ealls
Benson, Gall M	Sloux Falls
Berg, Sterling	Redfield
Berg, Tony L	Winner
Berg, Sterling	Rapid City
Berry, Jack T	Mitchell
Berry, Jack T. Bess, Michael A. Betts, Laurence S. Bhat, Dileep S. Bhatara, Vinod S. Bhatti, Tajammul H.	Sioux Falls
Betts Laurence S	Huron
Rhat Dileen S	Mitchell
Photogra Vined S	Ciany Falls
Dilatara, Villou S	Sloux Falls
Bnatti, Tajammui H	Sloux Falls
Bieberly, Frank G., Jr. Billion, John J. *Billion, Thomas J	Chamberlain
Billion, John J	Sioux Falls
*Billion, Thomas J	Sioux Falls
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Birkenkamp, Ray T	Mitchell
Blake, Jerome M	Sioux Falls
*Bloemendaal, Gerrit J	Ipswich
Bloemendaal, Robert D.	Rapid City
Blunck, Conrad F. J	Rapid City
	Sioux Falls
Boade, Werner, A	
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Brandenburg, Verdavne R.	Sioux Falls
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Brown, Bradley A	
Brown, Delbert L	Sioux Falls
Brown, Robert H Bruins, George S Brzica, Stephen M	. Aberdeen
Bruins, George S	Sioux Falls
Brzica, Stephen M	Sioux Falls
Buchanan, David J	Huron
Buchhammer, Carlos R.	Vermillion
Bucy, Christine F	Sioux Falls
Bunker, Thomas G	. Aberdeen
Burkhart, Thomas J	Sioux Falls
Burnap, Donald W	Rapid City
Burnett, Raymond G	Rapid City
Burns Edith A	Sioux Falls
Burns, Edith A Burns, Howard W	Sioux Falls
*Burns, Kendall R	Sioux Falls
Butz, Gerald W	Rapid City
Calhoon, Stephen L	Rapid City
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Listing
Carlson, Gary L
Dappen, Robert W. Mitchell Davis, John B. Pierre Daw, Edward F. Sioux Falls Dean, Roscoe E. Wessington Springs Dean, Thomas M. Wessington Springs DeClark, Robert P. Sioux Falls DeGeest, James H. Miller Delaney, Robert J. Mitchell Delaney, William A., Jr. Mitchell Dendinger, William J. Vermillion Desai, Bhasker J. Watertown Devick, John S. Colton Dewald, Allan L. Rapid City *Dickinson, John Canistota Dickson, Daryl S. Hot Springs Dilger, Joseph T. Mitchell Donahoe, John W. Sioux Falls Drummond, Ronald G. Rapid City Drymalski, Walter G. Sioux Falls D'Souza, Edward P. Aberdeen Duggan, Robert Pierre Dzintars, Paul F. Rapid City Dzintars, Valdis A. Sioux Falls
Easton, Jessie K. M Sioux Falls Ebbert, Larry P Rapid City

Eckrich, Jerome A., Jr Aberdeen	Hanson, Bernie H. P Watertown	Janis, John B Sioux Falls
Eckrich, Jerome A., Sr Aberdeen	Hanson, William O Huron	Janusz, Albin J Aberdeen
*Eirinberg, Isadore D Sioux Falls	Hare, Helen Jane Rapid City	Jaqua, Richard A Sioux Falls
Elk, Sidney A Rapid City	Harlow, Mark C Aberdeen	Javurek, Anthony J Sioux Falls
Elitary Mail I		Jenter, George W Sturgis
Elkjer, Neil J Sioux Falls	Harris, Russell H Rapid City	Jenier, George W Sturgis
Elson, David L Sioux Falls	Hart, Harvey J Aberdeen	Jentes, Paul K Fort Meade
Elston, John T Rapid City	Hartmann, Alfred E Sioux Falls	Jerde, O. Myron Rapid City
Engelhart, Kenneth E Watertown	Hartzell, Allan J Sioux Falls	Johnson, Beth L Sioux Falls
English, Gilbert L Sioux Falls	Haugan, Haakon O Rapid City	Johnson, Charles A Lemmon
Emphana Danamas I Signar Falls	Hayes, Robert H Wall	Johnson, Dennis L Sioux Falls
Ensberg, Dorence L Sioux Falls		
Epp, Dennis L Freeman	Hays, Laura M Mitchell	Johnson, Edward A Milbank
11,	Heidepriem, Glen Rapid City	Johnson, Robert C Sioux Falls
	Hainamann Dhyllia E Abardaan	Johnson, Robert K Rapid City
Fahrenwald, Myron E Aberdeen	Heinemann, Phyllis E Aberdeen	Johnson, Robert R Rapid City
Farrell, Harry W Sioux Falls	Heinrichs, Eberhard H Vermillion	Johnson, Thomas C Yankton
	Held, Gordon R Sioux Falls	Johnson, Virginia P Vermillion
Fedt, Donald N Watertown		
Felker, James R Sioux Falls	Henderson, Ben J Mobridge	Jones, John B Chamberlain
	Henrickson, Lynn A Sioux Falls	Jones, Warren L Sioux Falls
Fenton, Lawrence J Sioux Falls	Henrickson, Robert G Sioux Falls	Jones, William E Sturgis
Ferrell, Michael R Sioux Falls		
Ferrell, Robert L Rapid City	*Henry, Robert B Brookings	Judge, John O Mitchell
	Henry, Thomas E Tucson, AZ	Judge, Timothy J Mitchell
Finley, Richard C Rapid City		
Finney, Lawrence W Sioux Falls	Herbrandson, Clarence R Spearfish	Justice, Michael W Dell Rapids
*Fisk Pohert C Flandroom	Hercules, Costas Rapid City	
*Fisk, Robert G Flandreau	Hermann, Harland T Mitchell	W 11 FW P
Fletcher, Harold J Vermillion		Kalda, Ellison F Platte
Flohr, Charles E Mitchell	Hermanson, John M. Valley Springs	Kalda, Ellison F., II Sioux Falls
	Herrin, Gerald R Pierre	
Flom, Jon O Yankton	Heth, Samuel R Mitchell	Kangley, Daniel J Sioux Falls
Flora, George C Sioux Falls		Kapur, Hiroo R Huron
Foley, Robert J Tyndall	Hewitt, John M Rapid City	Kapur, Ravi, Huron
	Hockett, Richard D Mitchell	
Forshner, Reginald S Hot Springs	Hofer, Emil A Huron	Karlen, Louis W DeSmet
Foss, J. Frank Sioux Falls	noter, Enin A nuron	Kass, Joseph Rosholt
*Fox, Stanley W Canada	Hoffsten, Phillip E Pierre	Kaufman, Irvin I Freeman
	Hogrefe, Louis H Gregory	
Frazer, Paul D Clear Lake		Kazi, K. Stephen Aberdeen
Freeman, Jerome W Sioux Falls	Hohm, Byron T Sioux Falls	Kelley, Donald H Rapid City
	Hohm, Paul H Huron	
Freimark, Lyle G Rapid City	Hohm, Robert C Huron	Kelts, K. Alan Rapid City
*Friefeld, Saul Minneapolis, MN		Kemp, Earl D Sioux Falls
Friess, Richard W Sioux Falls	Hohm, Theodore A Huron	Kemp, James S Mitchell
Fromm, Harold E Rapid City	Holland, Lambert W Chamberlain	Kennelly, Daniel J Sioux Falls
	Holm, Richard P Brookings	
Frost, Donald M Sioux Falls		*Kershner, Calvin M Brookings
Frost, Harold L Rapid City	Holt, Bevley D Greeneville, TN	Kim, Thomas H Huron
Fuller, William C Sioux Falls	Holzwarth, David R Yankton	King, Bernard F Salem
Tulici, William C Sloux Tans	Honke, Richard W Wagner	
		*King, Lyndon M., Jr Sioux Falls
Gehring, Stephen H Sioux Falls	Honke, Richard W., II Wagner	Kittelson, H. Otis Sioux Falls
	Horner, William Sioux Falls	Klar, Werner Fort Meade
Gerber, Bernard C Aberdeen	Horning, James R Watertown	
Gere, Richard G Mitchell	Hosen, Richard S Sioux Falls	Knecht, John F Martin
Giebink, Robert R Sioux Falls		Knowles, Roy C Sioux Falls
	Hoskins, John H Sioux Falls	Knudson, Donald H Sioux Falls
*Gilbert, Freeman J Belle Fourche	Hoversten, David L Sioux Falls	
		Knutson, Dennis D Sioux Falls
	Houland Ismac I Abandaan	
Gill, Timothy J Rapid City	Hovland, James I Aberdeen	*Kohlmeyer, Frederick C. Sioux Falls
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell	Hovland, James I Aberdeen Howard, William J Rapid City	*Kohlmeyer, Frederick C. Sioux Falls
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish	Howard, William J Rapid City	Kom, Carlton J Aberdeen
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish	Howard, William J Rapid City Howe, Jerome K Mitchell	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish Goodhope, Robert C Fort Meade	Howard, William J Rapid City Howe, Jerome K Mitchell Hoxtell, Eugene O Sioux Falls	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish Goodhope, Robert C Fort Meade Graham, Donald B Sioux Falls	Howard, William J Rapid City Howe, Jerome K Mitchell	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H Rapid City
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish Goodhope, Robert C Fort Meade	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H Rapid City Kortum, Cynthia L Huron
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish Goodhope, Robert C Fort Meade Graham, Donald B Sioux Falls Grau, Thomas J Sioux Falls	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield Huber, Thomas J. Pierre	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H Rapid City Kortum, Cynthia L
Gill, Timothy J Rapid City Gillis, Floyd D., Jr Mitchell Golliher, Warren N Spearfish Goodhope, Robert C Fort Meade Graham, Donald B Sioux Falls Grau, Thomas J Sioux Falls Greenfield, Duane L Sioux Falls	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield Huber, Thomas J. Pierre Hubner, Jay W. Yankton	Kom, Carlton J Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H Rapid City Kortum, Cynthia L
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Gill, Timothy J	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield Huber, Thomas J. Pierre Hubner, Jay W. Yankton *Hubner, Roland F. Yankton Huet, William G. M. Huron Hughes, Howard D. Clear Lake Humphreys, Donald W. Sioux Falls *Huppler, Edward G. St. Paul, MN Hurley, Brian Sioux Falls Hurley, Timothy Sioux Falls Hussain, Rif'at Sioux Falls Hyland, Lowell J. Sioux Falls Ingvoldstad, James P. Sioux Falls Isburg, Carroll D. Yankton	Kom, Carlton J. Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H. Rapid City Kortum, Cynthia L. Huron Kosse, Karl H. Aberdeen Kovarik, Joseph A. Rapid City Kovarik, Richard A. Rapid City Kovarik, Wenzel J. Rapid City Krafka, Thomas L. Rapid City Kramer, Charles G. Chamberlain Kullbom, James B. Rapid City Kunz, James A. Rapid City Kurch, Julie Ann Huron Kwan, Francis P. Rapid City Lakstigala, Peters E. Sioux Falls Lampert, Arthur A., Jr. Madison *Lampert, Arthur A., Sr. Rapid City
Gill, Timothy J	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield Huber, Thomas J. Pierre Hubner, Jay W. Yankton *Hubner, Roland F. Yankton Huet, William G. M. Huron Hughes, Howard D. Clear Lake Humphreys, Donald W. Sioux Falls *Huppler, Edward G. St. Paul, MN Hurley, Brian Sioux Falls Hurley, Timothy Sioux Falls Hussain, Rif'at Sioux Falls Hyland, Lowell J. Sioux Falls Ingvoldstad, James P. Sioux Falls Isburg, Carroll D. Yankton Jackson, James W. Rapid City	Kom, Carlton J. Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H. Rapid City Kortum, Cynthia L. Huron Kosse, Karl H. Aberdeen Kovarik, Joseph A. Rapid City Kovarik, Richard A. Rapid City Kovarik, Wenzel J. Rapid City Krafka, Thomas L. Rapid City Kramer, Charles G. Chamberlain Kullbom, James B. Rapid City Kunz, James A. Rapid City Kunz, James A. Rapid City Kurch, Julie Ann Huron Kwan, Francis P. Rapid City Lakstigala, Peters E. Sioux Falls Lampert, Arthur A., Jr. Madison *Lampert, Arthur A., Sr. Rapid City Lang, Durward M. Omaha, NE
Gill, Timothy J	Howard, William J. Rapid City Howe, Jerome K. Mitchell Hoxtell, Eugene O. Sioux Falls Huber, Joel B. Redfield Huber, Thomas J. Pierre Hubner, Roland F. Yankton *Hubner, Roland F. Yankton Huet, William G. M. Huron Hughes, Howard D. Clear Lake Humphreys, Donald W. Sioux Falls *Huppler, Edward G. St. Paul, MN Hurley, Brian Sioux Falls Hurley, Timothy Sioux Falls Hussain, Rif'at Sioux Falls Hussain, Rif'at Sioux Falls Hushand, Lowell J. Sioux Falls Ingvoldstad, James P. Sioux Falls Isburg, Carroll D. Yankton Jackson, James W. Rapid City Jacobs, Tad B. Flandreau	Kom, Carlton J. Aberdeen Koob, K. Gene Sioux Falls *Koren, Paul H. Rapid City Kortum, Cynthia L. Huron Kosse, Karl H. Aberdeen Kovarik, Joseph A. Rapid City Kovarik, Richard A. Rapid City Kovarik, Wenzel J. Rapid City Krafka, Thomas L. Rapid City Kramer, Charles G. Chamberlain Kullbom, James B. Rapid City Kunz, James A. Rapid City Kurch, Julie Ann Huron Kwan, Francis P. Rapid City Lakstigala, Peters E. Sioux Falls Lampert, Arthur A., Jr. Madison *Lampert, Arthur A., Sr. Rapid City Lang, Durward M. Omaha, NE Lang, Terry A. Sioux Falls
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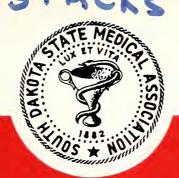
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Medicine

The Rural Family Medicine Clerkship (RFMC) at UNIVERGITY OF MARYLAND BALTIMORE the University of South Dakota School of Medicine: A Six Year Review SEP 29 '83

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The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

oral: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

NJECTABLE. To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used IV: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask If it is not feasible to administer Injectable Valium directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, i.e., phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effect. e amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to $2\frac{1}{2}$ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, futigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity;

insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

DIJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia. In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL-Adults: Anxiety disorders, relief of symptoms of anxiety—Valium (diaze-pam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose

Children: Tablets—1 to $2V_2$ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg daily when 5 mg oral Valium has been determined as the optimal daily dose (no for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg 1.M. or I.V. depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.) For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children administer I.V slowly; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals ut to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 10 mg (IV. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow IV. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful. In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatolog has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Vallium scored tablets — 2 mg, white: 5 mg, yellow; 10 mg, blue —bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules — 15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

NJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.



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S Medicine

The Rural Family Medicine Clerkship (RFMC) at the University of South Dakota School of Medicine: A Six Year Review 1977-1982

L. H. Amundson, M.D.* Burton W. Hancock, Ph.D.†

ABSTRACT

This paper presents a six year review of a community clinical clerkship model (RFMC) that is used at USDSM. It summarizes the experiences of 254 students who have taken this required senior clerkship and student evaluations offered by their clerkship instructors. This study also includes a six year review of student career choices by USDSM graduates. Although 25% of

There is a need for greater availability of more "primary care" to people and the medical manpower to deliver it. Assuming that it is a legitimate responsibility of a medical school to tailor its curriculum in such a way as to educate students appropriately for the needs of an area, a variety of programs must be available at the undergraduate medical education level. Currently in medical schools, most of the training is directed to hospital-based specialty or subspecialty care, while the majority of graduates will be providing medical care in an office or clinic setting. Since this care represents the bulk of care rendered, more attention should be paid to it

graduates entered family practice residencies, the data showed no clear cut relationship of ultimate career choice to this senior clerkship. The relationship of these results to family physician needs in South Dakota during the next decade, showing a need for 20 graduates to select family practice as a career, needs further study and delineation.²

in medical school education. It therefore appears that considerable ambulatory training for medical students is imperative in preparing these students for future health care delivery in America. Family medicine can offer the medical student of today experiences which not only augment the many biomedical and technical skills needed by physicians but also expand behavior, attitudes, and relationships with patients, families, communities, and peers.^{3, 4}

Therefore, during the clinical years it seems important to have block time exposures in family medicine while realizing that career choices have frequently been made by this time. These experiences often allow the student to "put it all together." For many students it is the first real life practice exposure outside of the medical center and allows the student for the first time a chance to put on "M.D. wings."

The ideal length of time for a block exposure in

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the last year of undergraduate medicine seems to be six to twelve weeks. A six week minimum, primarily assigned to one or two instructors, appears necessary as it takes two to three weeks for the student and the instructor to know and trust each other. Depending upon the site and the amount of clinical material available, twelve weeks may be too long if it is repetitious in a fairly confined area of exposure for the student. This, of course, will depend upon the curriculum of an individual school and the number of required and elective clerkships.

Methods

The RFMC at USDSM offers medical students the opportunity to provide continuing comprehensive care to patients over an eight week period during the senior year. At the time of this study, 254 graduates have taken this clerkship during a six year period and provide the cohort for this review.

This clerkship was modelled after the Community Clinical Clerkship in Family Medicine at the University of Washington,⁵ a clerkship whose clinical content and student experiences have been documented recently.⁶ Its structure and content have been described in earlier publications.^{1, 7}

The clinical clerkship goals used for this clerkship, which provide the basis for this study, can be summarized as follows:

- A. To provide the early basis of an educational continuum designed to train a family physician.
- B. To emphasize the importance of continuing,

- comprehensive patient care throughout the student's study of medicine.
- C. To provide a model within the medical school of family physicians providing continuing, comprehensive patient care, supplemented by specialty support.
- D. To relate these training experiences to the ultimate goal of providing proper family physician distribution by well trained, concerned, caring physicians.

Results

Student Experiences

Table I shows a composite of student experiences during this clerkship. Students saw an average of 63 patients per week in the office, worked up 25 hospital patients in the hospital during the clerkship, were able to see 22 patients two to three times during the RFMC, and saw nearly 7 patients more than four times. Their evaluations further showed that they did feel that they were part of the health care team.

Students felt that the strengths of the RFMC included: 1) responsibility for patient care, 2) excellent teaching, 3) a variety of patient problems, 4) office experience. They would have liked more experiences in the following areas during the course of their clerkship: 1) obstetrics, 2) minor surgery, 3) other office procedures, and 4) a greater exposure to the emergency room.

In the overall student evaluation of the RFMC experience, 96% of the students felt that this was an exceptional or good clinical experience (Table II).

NUMBER OF PATIENTS SEEN BY RFMC STUDENTS IN OFFICE												
patients seen	1977	1978	1979	1980	1981	1982						

Number of patients seen	1977	1978	1979	1980	1981	1982	Mean
Per week 2-3 times/RFMC More than 4 times/RFMC Number of hospital work-ups/RFMC	54	66	70	63	65	59	62.8
	16	19	27	22	29	29	22.2
	3	4	10	8	7	9	6.8
	23	23	27	28	28	25	25.7

TABLE II
USDSM
OVERALL STUDENT EVALUATION OF RFMC EXPERIENCE

	OVERALL STUDENT EVALUATION OF RFMC EXPERIENCE											
	1977	1978	1979	1980	1981	1982	Total					
Exceptional	22	33	27	42	32	32	188					
Good	12	3	10	2	18	12	57					
Adequate	2		1		2	1	6					
Marginal	2						2					
Unacceptable	1						1					
Total	39	36	38	44	52	45	254					

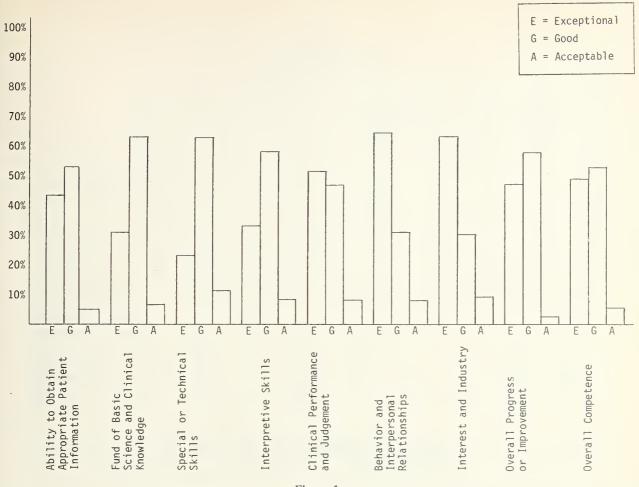


Figure 1
USDSM faculty evaluation of RFMC students. A six year summary 1977-1982.

	TABLE III USDSM GRADE DISTRIBUTION FOR RFMC SIX YEAR SUMMARY												
	1977	1978	1979	1980	1981	1982	Total	%					
A	22	26	28	31	24	20	151	59					
В	13	9	8	11	27	25	93	37					
C	4	0	2	2	1	0	9	4					
D	0	0	0	0	0	0	0	0					
F	0	1	0	0	0	0	1	0					
Total	39	36	38	44	52	45	254	100%					

Evaluation of the Student

Figure 1 shows a composite of faculty evaluations of the RFMC student during the first six years of this program. The data shows a large percentage of students receiving excellent, good, or average evaluations by faculty, using a nine item analysis instrument. These evaluations were done by faculty at 14 RFMC sites that have been used to date.

Figure 2 gives a comparison of RFMC student

evaluations by faculty on those students selecting family practice residencies and those selecting other specialties for graduate education. It can be seen that both student groups are similar in this three year comparison, especially in overall competence.

Table III shows the grading of students by this faculty composite. This being a "new" four year medical school, letter grades were required by LCME accreditation guidelines. Nearly two-thirds of the students received a letter grade A.

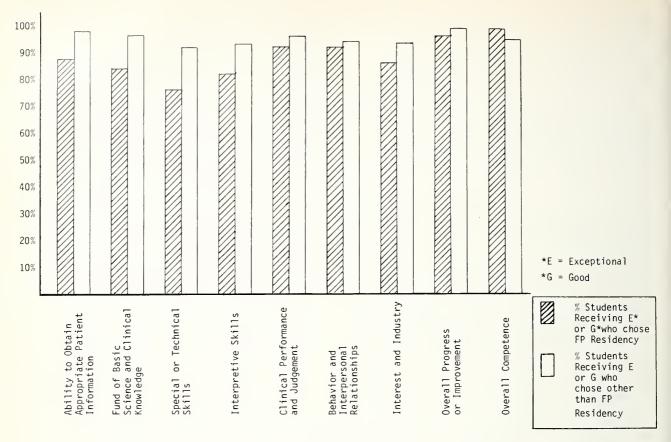


Figure 2 USDMS faculty evaluation of RFMC students. A three year comparison 1980-1982.

Residency Selection by Graduates

Several recent studies have looked at the impact of medical student experiences in family medicine on ultimate specialty selection. 8-15 Tables IV and V show the distribution of USDSM graduates in first year residencies and those entering family practice residencies by year of graduation. This data shows that 25% of USDSM graduates entered a family practice residency, compared to a 13% NRMP seven year average. The percentage of graduates entering transitional graduate programs is more than twice the national (NRMP) average. Other comparisons are similar.

Table VI shows the number of students taking their self-selected RFMC in sites of varying community size, and relates this to their selection of family practice as a career. Review of this data shows essentially no differences in career selection dependent only upon size of the RFMC community. However, this data does show one site in the less than 5,000 population category that has had 7 of 17 (41%) RFMC students select a family practice career, well above the school average of 25%.

Table VII compares those students entering fami-

ly practice residencies by third year clerkship base, showing a significant variation between the two main clerkship sites. This popular fourth year clerkship (RFMC) seems unable to overcome the third year clerkship base variable when selecting a career residency. ¹⁶

Table VIII gives a summary of specialty society student members and their selection of that specialty as a career choice. Whereas 55% of students voluntarily accepted student membership at the incoming third year level, only 28% of those entered a family practice residency. This figure compares with the total student selection (25%) of family practice as a career.

Table IX relates the time during the academic year that the RFMC was taken to the NRMP deadline and career selection. The data shows no demonstrable impact on career choice by timing of this required senior clerkship.

Discussion

This required senior clerkship has been a positive experience for medical students, clerkship instructors, and for the Department of Family Medicine at

TABLE IV DISTRIBUTION OF USDSM GRADUATES IN FIRST YEAR RESIDENCIES BY TYPE OF PROGRAM 1977-82 A SIX YEAR SUMMARY

	U.S. Graduates (NRMP 7 yr avg) 1976-82*	USDSM Graduates (6 yr summary) 1977-82			
Type of Program	Percent	Number	Percent		
Family Practice	13.0	64	25.0		
Internal Medicine	36.0	70	28.0		
Transitional (Flexible)	8.5	48	19.0		
Surgery	13.0	23	9.0		
Pediatrics	10.0	16	6.0		
Obstetrics/Gynecology	6.0	14	5.6		
Medical Specialties	4.5	4	1.5		
Dermatology					
Neurology					
Ophthalmology		(1)			
Psychiatry		(3)			
Surgical Specialties	2.5	8	3.1		
Neurosurgery					
Orthopedics		(6)			
Otolaryngology					
Urology		(2)			
Support Specialties	6.5	(2) 7	2.8		
Anesthesiology		(2)			
Pathology		(1)			
Physical Medicine					
Radiology		(4)			
Other					
	$\frac{100\%}{100\%}$	254†	100%		

^{*} Source: Journal of Medical Education, 54(4):347-349, 1979; 55(4):382-384, 1980; 56(4):372-374, 1981; 56(9):783-785, 1981; 57(5):349-428, 1982.

† 64 of 254 (25%) entered USDSM affiliated residencies.

TABLE V
USDSM M.D. GRADUATES ENTERING FAMILY PRACTICE RESIDENCIES
A SIX YEAR SUMMARY

Academic Year	Number of Graduates	Number Entering FP Residencies	Number Entering SD FPR Program
1976-77	39	9(23%)	4/9
1977-78	36	4(11.1%)	4/4
1978-79	39	11(28.2%)	7/11
1979-80	47	14(30%)	2/14
1980-81	49	20(41%)	8/20
1981-82	44	6(13.6%)	1/6
	254	64(25%)	26*/64(40%)

^{*} Six Residents left the program at the end of one year: 1 to enter another FP Residency, 4 to change discipline, 1 to enter practice.

the University of South Dakota School of Medicine.

Although 25% of USDSM students have chosen family practice as a career, a recently published study has shown that 20 students per year (40% of students graduating) should be entering family practice from this "family practice oriented school" to "relate these training experiences to the ultimate goal of providing proper family physician distribution by well trained, concerned, caring physicians."²

Although this study does not relate selection of a career in family practice to this senior clerkship in family medicine, based upon student and faculty evaluations, this type of educational exposure seems important for all medical students today, regardless of ultimate career selection. Other factors must be evaluated to help resolve the career choice issue at this "new" "family practice oriented" medical school.

TABLE VI USDSM STUDENTS CHOOSING FAMILY PRACTICE BY RFMC COMMUNITY SIZE*

RFMC Location	>10,000	5,000- 10,000	<5,000	% Total	% Total FP	% Site FP
Brookings	8/48%			19%	13%	17%
De Smet	0, 10 ,0		2/9	4%	3%	22%
Freeman/Bridgewater			0/5	2%	0%	0%
Gregory			7/17	7%	11%	41%
lot Springs			2/8	3%	3%	25%
Lead		6/20		8%	9%	30%
Madison		8/25		10%	13%	32%
Mobridge		5/15		6%	8%	33%
Pierre	8/35			14%	13%	23%
Rapid City	9/33			13%	14%	27%
Tyndall	5100		5/21	8%	8%	24%
Vermillion		0/2		1%	0%	0%
Watertown	4/13			5%	6%	30%
Wessington Springs	10		0/3	1%	0%	0%
Total RFMC						
Experience (N = 254)	129	62	63			
Fotal FP (N = 64)	29	19	16			
% Choosing FP	23%	31%	25%			

^{*} Number choosing family practice/number of students over six years.

TABLE VII
USDSM M.D. GRADUATES ENTERING FAMILY PRACTICE RESIDENCIES
BY THIRD YEAR CLERKSHIP BASE

		Third Year Clerkship Base*				
Academic Year	Number of Graduates	X (66%)	Y (34%)			
1976-77	39	8/26 (30.7%)	1/13 (7.6%)			
1977-78	36	4/23 (17.3%)	0/13 (0%)			
1978-79	39	9/27 (33.3%)	2/12 (16.6%)			
1979-80	47	11/31 (35.4%)	3/16 (18.7%)			
1980-81	49	16/33 (48.5%)	4/16 (25%)			
1981-82	44	5/29 (17.2%)	1/15 (6.6%)			
		53/169 (31.6%)	11.85 (13%)			
	254	64/254	(25%)			

^{*} Students are distributed 2/3 and 1/3 to clerkship sites X & Y, but self-select which site they prefer.

TABLE VIII
USDSM M.D. GRADUATES
AAFP (SPECIALTY SOCIETY)
STUDENT MEMBERSHIP

Academic Year	Number of Graduates	Number of Student Members	Number Entering FP Residencies	Number of Student Members Entering FP Residencies
1976-77	39	24	9 (23%)	7
1977-78	36	23	4 (11.1%)	4
1978-79	39	21	11 (28.2%)	8
1979-80	47	25	14 (30%)	8
1980-81	49	26	20 (41%)	12
1981-82	44	22	6 (13.6%)	1
	254	141 (55%)	64 (25%)	40/141 (28%)

TABLE IX USDSM MONTH(S) RFMC BEGAN (FP/TOTAL)

Year	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Sub- Total		Jan.	Feb.	Mar.	Apr.	May	Sub- Total	Total
1977 1978 1979 1980 1981 1982		2/4 4/7 1/7 2/14	0/5 1/6	0/6 0/7 3/6 4/8 7/9	0/1 2/16	1/7 0/7 1/7 3/8 3/10	1/1	1/18 1/20 7/19 11/23 11/26 6/45	NRMP Deadline	3/5 2/6 2/9 0/8 4/12	1/8 1/4	4/8 0/6 2/10 3/13 5/14			8/21 3/16 4/19 3/21 9/26 0/0	9/39 4/36 11/38 14/44 20/52 6/45
Total		9/32	3/26	14/36	2/17	8/39	1/1	37/151		11/40	2/12	14/51			27/103	64/254
Percen	t select	ing fan	nily pra	actice				24%							26%	25%

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S President's Page

The Social Security Amendments of 1983 (P.L. 98-21), signed into law on April 20, 1983, established a prospective payment system (PPS) for hospitals. As a result, Medicare payment will, in general, be based on a fixed, predetermined amount for each case. Classification into one of 467 diagnosis related groups (DRGs) will be based on the discharge diagnosis. Physicians' fees under Part B of Medicare, capital costs, medical education and skilled nursing facilities are not included under PPS. Congress has mandated the Secretary of Health and Human Services (HHS) to study and report on the advisability of including these items. The study on physician fees is to be reported by 1985. Final regulations, including the DRGs will be published by the Health Care Financing Administration (HFCA) by September 1, 1983. October 1, 1983 is the effective date for implementation of the PPS.

Nothing has had the impact on private practice of medicine that PPS and DRGs portend, since Medicare and Medicaid were established by law in 1966. Physicians cautioned government that a program of such enormity demanded careful implementation, threatened astronomical costs, and a demand for health services that could exceed the available resources. It was also pointed out that the proposed underwriting was inadequate to support the cost of the program. In less than twenty years Medicare is overused and in financial trouble. A private insurance program would be declared insolvent in the same situation.

Now one senses deja vu. Medicine cautions goverment that implementation of P.L. 98-21 is being hurried. Although Maryland and New Jersey have had a few years of experience with PPS, they have not learned enough to predict their futures with the program. Nonetheless the entire nation is being hurried into the same situation. Implications for diminishing the quality of health care for involved patients are real. The thrust seems to be toward the cheapest not the best care. Words, strange to American physicians, are being heard, e.g. "care will have to be rationed" or "allocated"; by whom, no one knows. My surmise is that the physician will "ration" service to the needy after funds are exhausted — for free. So what else is new? The voice of Medicine is rejected as it was before 1966.

P.L. 98-21 mandates a Prospective Payment Assessment Commission to advise the Congress and the Secretary of HHS on important aspects of the



new payment system. Included are the annual inflation factor, incorporation of new technology and treatment protocols, medical apropriateness of health resource use, and the efficiency of new and existing medical and surgical procedures. Fifteen experts in aspects of health care, e.g. physicians, nurses, employers, third-party payors and researchers are to comprise the commission. They should be objective as they monitor the changes evoked by PPS and DRGs, to ensure that Medicare patients get high-quality care, and that the fiscal integrity of hospitals is not impaired. Without the Commission, the Department of HHS would be payor, regulator and evaluator; a potential for concern. The fifteen members have not been named.

At the AMA meeting in June of 1983, the House of Delegates acted to promote input by hospital staffs into the DRG process to ensure that quality care is not compromised, and directed the Board of Trustees to vigorously pursue a program of education and assistance to the membership concerning DRGs.

Joseph N. Hamm, M.D. President

Joseph N. Hamm, M.D., President South Dakota State Medical Association

S Department of Health

Is a Statewide Tumor Registry Needed for South Dakota?

In June 1982, a local pathologist wrote to the South Dakota Health Department (SDHD) stating that over a short period in time six patients had been seen with transitional cell carcinoma of the ureter (an uncommon tumor), by a urologist whose service area encompasses about 150,000 people. Epidemiological information was requested in an effort to find possible environmental or occupational causative factors. Unfortunately, information available through the Center for Health Statistics of the SDHD was confined to mortality data, limiting its usefulness to those who were attempting to investigate the situation. Similar circumstances involving other malignancies have occurred on several occasions in various portions of this state during the past few years.

Cancer is one of the three great killers in the U.S. and in South Dakota today. With the anticipated increase in the aged population in this state in the next few years, the frequency of this disease can be expected to maintain its present level, or to increase. Although today excellent epidemiological data relating to cancer are available for the U.S., accurate information concerning the mortality and morbidity caused by cancer is incomplete for South Dakota. Only through carefully controlled epidemiological studies can environmental and occupational factors be identified and evaluated for their carcinogenic effect. To do such analyses, accurate population data are required.

Valuable information from the U.S. relating to the mortality and morbidity associated with cancer has been compiled and reported periodically by the American Cancer Society, and in statistical bulletins put out by some life insurance companies. Other studies have approached cancer from the epidemiological perspective, attempting to identify occupational or other hazards which may be causative or predisposing factors in this dread disease. 3-7

Mason and co-workers prepared a two volume atlas of cancer mortality for the U.S. 1950-1969. Information from the contiguous 48 states was obtained from death certificates which listed cancer as the cause of death. Deaths were compiled by the county of usual residence as listed on the certificates. Information obtained was plotted on maps of

the U.S. using color coding to indicate the frequency in various regions. Although these maps provided interesting information upon which to speculate, demographic analyses utilizing death certificate information as the reference source, provided data of questionable value to statisticians. ¹⁰ In addition, only cancer mortality was evaluated. The studies did not reflect morbidity, or changes in morbidity which might indicate the effectiveness of anti-cancer therapy.

South Dakota's first tumor registry was started at Yankton in the 1950s. Subsequently, tumor registries have been developed in Sioux Falls, Aberdeen, Watertown, and Rapid City. To date the tumor registries activities have been largely those of data storage, with minimal effort to compile data or to trace demographic patterns. Some tumor data from South Dakota hospitals are now stored in repositories located outside this state. The multiplicity of repositories for cancer data storage increases the difficulty of assembling demographic information concerning cancer and hinders the identification of "cancer hot spots" in South Dakota.

On May 26, 1983, at a meeting sponsored by the Rapid City Regional Hospital, Inc., the formation of a statewide tumor registrars association was proposed. Medical record office personnel from all parts of the state were invited. Because vital statistics are an integral function of the SDHD, cancer mortality data are an important part of death records and statistics, and information relating to morbidity from cancer is sadly deficient for this state, the SDHD was represented at the organizational meeting

The possibility exists that if a statewide cancer registry can be made operational, the computer capabilities of the SDHD can be utilized to centralize and process cancer related data. To be effective, input into the system will be necessary from as many patient care facilities, as possible. Standard terminology, standard data entry techniques, and a good measure of cooperation between and among the participants in a cancer registry will be necessary to facilitate data processing and retrieval. Periodic demographic reports can be made by the SDHD relating to the types of cancers found, their anatomic locations, and their geographic representation within South Dakota.

John B. Gregg, M.D.
Willis F. Stanage, M.D.
Lawrence J. Massa
Office of Medical Services
South Dakota Department of Health

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Minnesota Medical Association Resource Group on **Rheumatic Diseases**

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RHEUMATOLOGY SEMINAR V March 6-March 13, 1984

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Announcement

Dr. Judson O. Mabee is pleased to announce the association of Ray T. Birkenkamp, M.D. in the practice of ophthalmology in Mitchell, SD.

Dr. Birkenkamp is originally from Litchfield, IL, and received his undergraduate degree from the Univ. of Illinois. He received his medical degree from Southern Illinois Univ., and did a general internship at Lutheran Medical Center in St. Louis, MO. He then served for two years in the U.S. Public Health Service in Wagner, SD, and has now returned to South Dakota following a residency in ophthalmology at St. Louis Univ. Hospitals, St. Louis, MO.

Dr. Birkenkamp will practice general ophthalmology.

S Chapter News

R for the 80s





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

FUTURE DIRECTIONS FOR MEDICAL EDUCATION

A Report of the Council on Medical Education Adopted June 15, 1982, by the House of Delegates of the American Medical Association

Part III

Recommendation 29:

The medical profession should continue to encourage participation in continuing medical education related to the physician's professional needs and activities. Efforts to evaluate the effectiveness of such education should be continued.

Recommendation 30:

The medical profession and the public should recognize the difficulties related to an objective and valid assessment of clinical performance. Research efforts to improve existing methods of evaluation and to develop new methods having an acceptable degree of reliability and validity should be supported.

Recommendation 31:

United States citizens should have access to factual information on the requirements for licensure and for reciprocity in the various jurisdictions, prerequisites for entry into graduate medical education programs, and other factors that should be considered before deciding to undertake the study of medicine in schools not accredited by the Liaison Committee on Medical Education.

Recommendation 32:

Policies governing the accreditation of U.S. medical education programs specify that core clinical training be provided by the parent medical school; consequently, the AMA strongly objects to the practice of substituting clinical experiences provided by U.S. institutions for core clinical curriculum of foreign medical schools. Moreover, it strongly disapproves of the placement of any medical school undergraduate student in hospitals and other medical care delivery facilities that lack educational resources and experience for supervised teaching of clinical medicine.

Recommendation 33:

Methods currently being used to evaluate the readiness of graduates of foreign medical schools to enter accredited programs in graduate medical education in this country should be critically reviewed and modified as necessary.

No graduate of any medical school should be admitted to or continued in a residency program if his or her participation can reasonably be expected to affect adversely the quality of patient care or to jeopardize the quality of the educational experiences of other residents or of students in educational programs within the hospital.

Recommendation 34:

The Educational Commission for Foreign Medical Graduates should be encouraged to study the feasibility of including in its procedures for certification of graduates of foreign medical schools a period of observation adequate for the evaluation of clinical skills and the application of knowledge to clinical problems.

Recommendation 35:

The American Medical Association, in cooperation with others, should continue to review and define standards for medical education at all levels. The AMA should continue to participate

in the evaluation and accreditation of medical education at all levels.

Recommendation 36:

The American Medical Association, when appropriate, should use selected consultants from the public and from the professions for consideration of special issues related to medical education.

EPILOGUE

This report has addressed various forces, past and present, which have had a significant influence upon the education of physicians. Some of the current and future problems of medical education have been identified, and recommendations for their resolution have been presented.

Medical schools, teaching hospitals, and professional organizations have a considerable degree of freedom to define and conduct their educational programs within the general principles established by the respective accrediting bodies. Institutional objectives differ, and this diversity is a desirable attribute as long as the goals and objectives of educational programs are clearly delineated and the basic tenets of excellent education are adhered to.

As stated at the beginning of this report, no attempt has been made to address all of the problems confronting medical education in the foreseeable future. Among those not considered in depth but in need of study by the medical profession, medical educators, and the public are:

- 1. Cost and financing of medical education;
- Changing ethical principles resulting from new knowledge and technology;
- Interrelationships between governmental agencies and higher education;
- 4. Relationship of specialty board certification to licensure;
- 5. Expectations of society concerning physician competence;
- Methodologies for evaluating clinical performance; andNeed for cohesive long-range planning nationally for all

levels of medical education. PATIENT EDUCATION TIPS

Reducing Your Waistline And Your Blood Pressure

Overweight? Some patients may not know it, but shedding those unwanted pounds can do more than shrink the waistline. It might also lower the blood pressure.

High blood pressure is twice as common among people who are overweight. When these patients take pounds off and keep them off, chances are they'll need less medicine (or none) to lower pressure to a healthy level. And many people with normal blood pressure can help keep it that way by watching their weight.

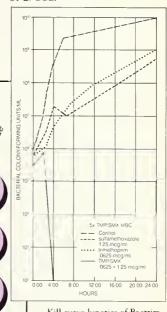
Advise your patients about keeping their weight and blood pressure under control.



Bactericidal activity

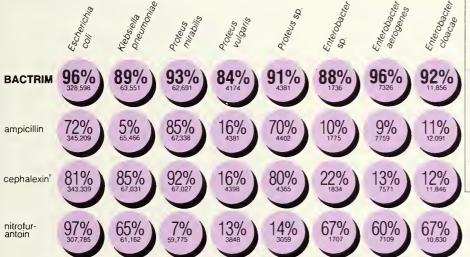
with minimal resistance

RAPID IN VITRO DESTRUCTION OF E. COLI*



Kill curve kinetics of Bactrim and its individual components against E. coli in vitro. ¹

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials



[†]Analogous to cephalothin, the primary antibiotic disc used in testing.

Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.

Numbers under percentages refer to the projected number of isolates tested.

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*. ¹² and on clinical isolates of *E. coli*, *Klebsiella-Enterobacter*, *Proteus mirabilis* and *Morganella morganii*³—the most common causative organisms of urinary tract infections. ⁴ More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms. ⁵ In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy. ⁶⁻¹¹

Resistance to Bactrim develops more slowly than to either of its components alone in vitro.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.^{5,12} Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. for recurrent urinary tract infections

*In vitro data do not necessarily predict clinical results.

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Bactrim DS (trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

which follows: Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. Note: The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of Haemophilus influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

safety of repeated use or bactum in chinator and indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of Haemophilus influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over a single antimicrobial agent. For enteritis due to susceptible strains of Shigella flexneri and Shigella sonnei when antibacterial therapy is indicated. Also for the treatment of documented Pneumocystis carinii pneumonitis. Contraindications: Hypersensitivity to trimethoprim or sulfonamides, patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age. Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with Immethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

be early signs of serious blood disorders. Frequent CBCs are recommender, therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with lotic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Allergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. Gastrointestimal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colisis and pancreatitis. CNS reactions: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, finnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. Miscellaneous reactions: Drug lever, chilis, toxic nephrosis with oligoric agents,

ussage for 5 days for snigellosis. Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis. For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is believe 15 ml/min.

below 15 ml/min.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS. Usual adult dosage. 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days. PNEUMOCYSTIS CARINII PNEUMONITIS:

PNEUMÓCYSŤIS CARINII PNEUMONITIS:
Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100, Prescription Paks of 20. Tablets, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. Pediatirc Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oc (1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).



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BRIEF SUMMARY PROCARDIA* (nifedipine) CAPSULES

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCAROIA (infedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm proto aright at rest accompanied by 31 segment relevation, 2 aligned to formary aftery spasm in those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PMULAHOLIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCAROIA is indicated for

the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCAROIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are

but confirmation of sustained effectiveness and evaluation or long-term salety in those patients are incomplete. Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent freatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.) CONTRAINDICATIONS. Known hypersensitivity reaction to PROCARDIA WARNINGS. Excessive Hypotension. Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients screining PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with often racrotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PRDCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawa!: Patients recently withdrawn from beta blockers may develop a with-Severe hypotension and/or increased fluid volume requirements have been reported in patients

Resulting from increased near rate alone. Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCAROIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather fhan stopping them abruptly before beginning PROCAROIA. PROCAROIA

Congestive Hearf Failure: Rarely, palients, usually receiving a beta blocker, have developed hearf failure after beginning PROCAROIA Patients with tight aortic stenosis may be at greater risk for

failure after beginning PROCARÖIA Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PRDCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodiation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCAROIA. This edema occurs primarily in the lower extremities and usually responds to diuretic herapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug inferactions: Beta-adrenergic blocking agents is usually well tolerated, but there have been occasional interature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCAROIA may be safely co-administered with infrates, but there have been in controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis Administration of PROCAROIA may be safely co-administered with infrates, but there have been incontrolled studies to evaluate the antianginal effectiveness of this combination.

Digitalis Administration of PROCAROIA may be safely co-administered with infrates, but there have been incontrolled study of over two houndred patients with coronary artery disease. In an uncontrolled study of over two houndred patients with coronary artery disease In an uncontrolled study of over two houndred patients with coronary artery disease In an uncontrolled study of over two houndred patients with coronary artery disease In an uncontrolled study

dipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose
Pregnancy: Category C. Please see tull prescribing information with reference to teratogenicity in
rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.
ADVERSE REACTIDNS: The most common adverse events include dizziness or light-headedness,
peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patents, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5% of
Syncopal episodes did not recur with reduction in the dose of PROCAROIA or concomitant antianginal medication. Additionally, the following have been reported muscle cramps, nervousness,
dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joinf stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, printisu, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCAROIA therapy was
associated with an increase in anginal pain, possibly due to associated hypotension.
In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of
these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase. CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevaled transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder
disease after about eleven months of infedigine therapy. The relationship to PROCAROIA therapy is
uncertain. These laboratory abnormalities have rarely been associated with cl

INTERTURE

HOW SUPPLIED: Each orange, soff gelatin PROCAROIA CAPSULE contains 10 mg of infedipine

PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NOC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59 10 77°F (15° to 25°C) in the manufacturer's original container

More detailed professional information available on request

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"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,¹ taking fewer nitroglycerin tablets,² doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

*Procardia is indicated for the management of:

1) Confirmed vasospastic angina.

2) Angina where the clinical presentation suggests a possible

vasospastic component.

3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.



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S Clinicopathologic Conference

Seventy-Four Year Old Caucasian Female with Filling Defect in Cecum by X-ray and Blood Loss Anemia

Margaret R. Devick, M.D.*
Dorence L. Ensberg, M.D.†
Discussers

John F. Barlow, M.D.‡ Editor

Case #M973 103

This 74-year-old caucasian female entered Sioux Valley Hospital for evaluation of a probable lesion in the region of the cecum.

The patient first noted symptoms approximately three months prior to admission with the onset of weakness and shortness of breath so that she was unable to walk up a flight of stairs without dyspnea and considerable fatigue. She saw a local physician who obtained a hematocrit of 24 vol./dl. The patient was treated with oral iron and subsequently parenteral iron therapy when she did not tolerate the oral iron therapy. The hematocrit rose to the level of 37 to 39 vol./dl. Associated with the weakness the patient had episodes of crampy midabdominal pain, associated with bloating, and firmness in her abdomen. She did not admit to a change in her bowel habits and noted no gross blood in the stool or change in stool color until she took the iron therapy. The patient did have hemorrhoids, and attributed occasional bright red blood in the stool to that cause. The patient had a history of chronic constipation prior to her onset of symptoms but had had no diarrhea.

Two days prior to admission, the patient had had a barium enema and a filling defect near the ileocecal valve was found. There was no familial history of colon cancer.

The patient had no significant history of other hospitalizations or illnesses. She had seven children and was on oral medication for hypertension, which was under control. During her recent workup, an asymptomatic urinary tract infection had been discovered. During recent episodes of abdominal pain, the patient was not able to urinate but had no dysuria, frequency, or urgency

PHYSICAL EXAMINATION: Height 155 cm., weight 76 kg., temperature 37°C, pulse 64/min. and regular, respirations 18/min. and regular, blood pressure 170 systolic and 80 diastolic. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The heart was not enlarged and there were no murmurs or extra sounds. Examination of the breasts was unremarkahle. The abdomen was soft, flat and nontender. There were no palpahle organs or masses. There was no tenderness or spasm. There was a small umbilical protrusion and diastasis recti. The patient had external and mildly prolapsed internal hemorrhoids. There were no masses by rectal examination. Examination of the extremities revealed osteoarthritic changes in the knees. The pulses were bounding bilaterally. LABORATORY DATA: Urinalysis yellow, clear, specific gravity 1.012, pH7.0, negative for protein, glucose, ketone bodies, hile, hemoglohin; sediment negative. Hemoglobin 13.9 gm/dl, hematocrit 43 vol/dl, mean corpuscular hemoglobin 26 pg; mean corpuscular volume 81 fl, mean corpuscular hemoglobin concentration 32%, total leukocyte count $4{,}700/\text{mm}^3$ (4.7 × $10^9/\text{L}$) with 54% segmented neutrophils, 6% neutrophilic bands, 3% eosinophils, 37% lymphocytes. The red cells were slightly hypochromic microcytic on smear. Platelet count $352,000/\text{mm}^3$ ($352 \times 10^9/\text{L}$), sodium 137 meq/L, potassium 3.7 meq/L. A 12-panel chemistry study was within normal limits except for a total protein of 4.8 gm/dl, albumin of 2.7 gm/dl with a nondiagnostic electrophoretic pattern. A chest film showed horderline cardiomegaly with a prominent tortuous aorta. The heart and lungs were otherwise normal. An electrocardiogram showed ST and T wave changes consistent with anteroseptal ische-

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DR. DEVICK: I would like to make a few comments before I discuss the differential diagnosis of this woman's cecal lesion. Unless an etiology for an anemia is already known, any person who presents to a physician with an anemia should have an evaluation to find the etiology of the anemia prior to therapy. Any person over 50 years of age who presents with anemia should certainly have tests for occult blood in the stool and an investigation of the gastrointestinal tract for neoplasm.

I am not sure from the history whether this woman had an evaluation of anemia prior to the initiation of iron therapy. Indeed, the anemia in this case certainly seems to be occult blood loss from gastrointestinal bleeding.

I would like you to note the barium enema study which shows a smooth oval defect in the cecal area (Figure 1).

I have divided the possible causes of this cecal lesion into four categories — inflammatory lesions, benign tumors, malignant tumors, and miscellaneous conditions.



Figure 1 Spot film of filling defect in the cecum.

Inflammatory conditions: There are few inflammatory lesions of the bowel consistent with this woman's history of possible gastrointestinal bleeding, chronic constipation, occasional crampy abdominal pain and absence of significant findings on abdominal physical examination.

A solitary cecal ulcer is a possibility since it may present with chronic intermittent pain, constipation and occult blood loss. However, the pain is usually in the right lower quadrant and the patient usually has a fever as well as tenderness or a tender mass in the right lower quadrant. The finding on barium enema examination is that of an ulcer rather than a smooth oval filling defect as in this case. One must

also consider tuberculosis, amebiasis, and Crohn's disease, but these inflammatory conditions usually have a more distinct history and other ulcerations or abnormalities on a barium study.

Actinomycosis in its abdominal form is uncommon but the x-ray may be mistaken for a carcinoma of the cecum, giving rise to an irregular mass and symptoms suggesting an appendiceal abscess.

Benign tumors: Adenomatous polyps in varying sizes and shapes are very common lesions of the colon. They may be found throughout the entire large bowel, even though they tend to be more frequent in the sigmoid colon and rectum. They are unusual before the age of 20 but show a progressive increase in incidence until about the 8th decade of life when they are found in almost 50% of the population. Adenomatous polyps seldom produce symptoms until they reach considerable size when occult bleeding may occur. Pain accompanying the lesion is not common and is usually due to traction on the lesion by the peristalsis of the bowel creating an intussusception.

Villous or papillary adenoma is quite an unlikely candidate for the lesion in this case as the tumor is often broad-based and slightly irregular and unlike this polypoid smooth mass. Patients with large lesions often have diarrhea with mucus and electrolyte imbalance but occult bleeding causing anemia is rather uncommon.

Lipomas are common benign tumors and may be seen anywhere throughout the bowel. The right colon is the most common area in which these tumors are found. They originate in the submucosa and are usually sessile, smooth and round. They can present a defect on barium enema which is not always constant. Painless, occult blood loss may occur. Recurrent abdominal pain may occur as a result of intussusception as described above.

A leiomyoma is a benign intramural tumor composed of smooth muscle which arises from the bowel wall and usually remains asymptomatic until they reach large size. Ulceration may occur with subsequent blood loss.

A hamartoma is a benign tumor composed of an abnormal mixture of normal tissue elements. One example is the so-called juvenile polyp. They usually occur in a younger age group, may be found singly or multiply and may present with occult rectal bleeding or abdominal pain.

Neurofibroma, lymphangioma and hemangioma are other intramural benign tumors which may occur and cause gastrointestinal bleeding and abdominal pain but are all quite uncommon.

Malignant tumors: Adenocarcinoma of the colon frequently affects females and has a peak inci-

dence in the fifth through the seventh decade. Only 15% of all carcinomas of the colon are found in the cecum and ascending colon, although this number has been much higher in recent studies. Adenocarcinomas occurring in the right colon are often polypoid but not smooth, but symptoms of anemia with fatigue, weakness and shortness of breath as well as weight loss occur in 25-50% of the patients. Diarrhea is a rare symptom in cecal lesions but constipation may be present. Abdominal pain is a fairly common complaint. Of course, adenocarcinoma is the most likely diagnosis in this case and is the one that must be ruled out before any other possibility is considered.

Carcinoid tumor is a slow growing malignant tumor arising from the enterochromaffin cells in the crypts of Lieberkuhn. They are very common in appendix as an incidental finding. Less than 20% of these lesions arise in the cecum, but they may occur anywhere in the gastrointestinal tract, most commonly in the ileum. These lesions may cause symptoms similar to other tumors of the gastrointestinal tract but also may excrete serotonin when they metastasize to the liver and may be accompanied by carcinoid syndrome with cutaneous flushing, diarrhea and abdominal cramping. Intestinal bleeding and obstruction can occur. The tumors are usually infiltrative rather than polypoid.

Leiomyosarcoma is usually a large bulky malignant tumor derived from smooth muscle which may produce bleeding, obstruction or perforation. Constipation and abdominal pain may occur.

Malignant lymphomas may arise in the cecum and infiltrate the bowel wall but may also present as polypoid tumors with abdominal pain and gastrointestinal bleeding. A localized primary lymphoma of the gastrointestinal tract usually occurs in patients over fifty and may be associated with abdominal pain as well as weight loss, nausea, vomiting, anorexia and anemia.

Lastly, metastatic carcinoma to the cecum may occur and present as a polypoid lesion. Malignant melanoma particularly may present in this manner but is usually not a single lesion. We have no history of a primary malignant tumor at another site in this patient.

Miscellaneous conditions: The ileocecal valve itself may be seen on barium enema examination. It is only seldom mistaken for a mass. However, lipomatous infiltration of the ileocecal valve due to submucosal infiltration of adipose tissue may present as a filling defect which appears as a smooth mass or masses on barium enema examination. This lesion is a distinct possibility in this case and may be associated with bleeding.

Edema of the ileocecal valve region from intermittent intussusception is another possibility in this case.

A mucocele of the appendix may occur when there is proliferation of mucus-secreting epithelium within the appendix producing distention of the appendiceal lumen. This may cause a filling defect in the cecum but these are usually incidental findings at surgery or autopsy. It could cause a globular mass in the cecum on barium enema.

An appendiceal stump may be mistaken for a cecal mass but this is usually smaller than the lesion in this case and rarely causes symptoms. Primary appendiceal intussusception is a rare cause of a filling defect in the cecum which may be round or oval, and may present acutely or give rise to intermittent symptoms.

Solitary cecal diverticum is thought to be congenital and is usually a chance finding, although acute diverticulitis may occur.

Acute appendicitis or Crohn's disease could cause a mass such as this due to an abscess.

A fecalith or fecaloma may be found in elderly patients who have chronic constipation but the lesion should have no attachment to the mucosal surface.

A so-called eosinophilic granuoloma or inflammatory polyp of the bowel may occur in this location but is usually seen as a pedunculated or sessile lesion elsewhere in the intestinal tract, particularly the stomach.

An extrinsic lesion could produce this defect. A benign or malignant extrinsic tumor of the ovary could be responsible. Any extrinsic malignant tumors could certainly infiltrate the bowel wall. Any lesion arising from the adjacent soft tissue could produce a tumor with a defect on barium enema examination.

Malakoplakia is a poorly understood condition which may be associated with malignancy of the colon or other conditions. It is a granulomatous disease of unknown cause which usually affects the urinary tract but has been seen in the large bowel and other organs. It has been associated with fever and gastrointestinal bleeding. The defect may present on barium enema or on colonoscopy as an irregular, ulcerated mass.

Dr. Margaret Devick's diagnosis: Mass in cecum Rule out adenocarcinoma of the colon

DR. BARLOW: We received a 27 cm. length of bowel including the terminal, ileum, ileocecal valve and ascending colon. In the region of the ileocecal valve were many polypoid soft yellowish masses grossly appearing like adipose tissue. Microscopic examination showed an intact mucosa beneath

which there was extensive growth of mature adipose tissue which had proliferated and given rise to the polypoid structures in the ileocecal valve region (Figure 2). Even after careful search, an ulceration accounting for the source of the bleeding could not be found, but an ulcer may have healed subsequent to the patient's episodes of bleeding.



Figure 2
Note intact mucosa overlying adipose tissue and smooth muscle of bowel wall.

This condition is called lipomatosis of the ileocecal valve. It should be distinguished from the encapsulated circumscribed lipoma which may occur anywhere throughout the gastrointestinal tract. This lipomatous transformation of the ileocecal valve has also been called pouting ileum, hypertrophy of the ileocecal valve, fatty degeneration of the ileocecal valve, submucosal fatty accumulation or lipohyperplasia of the ileocecal valve. The condition is rare and the etiology is unknown. It is more common in females, but the patients need not be obese. Most cases occur between the ages of 40 and 70 years of age. The condition may be asymptomatic but obstructive symptoms as well as acute and chronic blood loss have been described. Resection seems to be the therapy of choice.

FINAL ANATOMIC DIAGNOSIS: LIPOMATOSIS OF THE ILEOCECAL VALVE

DR. ENSBERG: At surgery, the patient had a soft spongy mass in the region of cecum on palpation. Because I could not determine its nature, I performed a right hemicolectomy removing a portion of the terminal ileum, ileocecal valve as well as the ascending and right transverse colon. This is a typi-

cal operation for carcinoma, but I do only a limited resection of the cecum for Crohn's disease.

Lipomatosis of the ileocecal valve can present with bleeding or obstructive symptoms. The obstructive symptoms are due to intermittent intussusception of the lesion. As noted, lipomas may occur anywhere throughout the bowel and I have seen cases presenting with massive hemorrhage. I believe one would have to consider this a case of adenocarcinoma of the colon until proved otherwise and have treated it accordingly. I believe it is interesting that there has been a shift to the right in the location of carcinoma of the colon. In other words, we see more right sided lesions, which traditionally present as anemia, than we did in former years.

DR. WARREN JONES:* I would like to emphasize that you must keep a high index of suspicion for carcinoma of the colon and cecum. If any filling defect of the cecum is found, one must strongly suggest operation. Even if an inconstant defect on barium enema is noted by the radiologist, I would suggest a study. Adenocarcinoma of the bowel is such a frequent lesion that no opportunity to diagnose it at an early stage can be missed.

DR. DANIEL HEINEMANN:† What do you think the routine screening examination for carcinoma of the colon should be?

DR. ENSBERG: It used to be said that anyone over the age of 50 should have a proctoscopic examination every year. I believe the suggestion is that these examinations can be less frequent at the present time. I believe that testing for occult blood in the stool is helpful and a history is very important. A change in bowel habits or a history of the presence of blood in the stool are very ominous. Of course, the presence of anemia or chronic blood loss should always trigger a possible evaluation for carcinoma of the colon.

DR. DEVICK: What would you do if the patient had a positive occult blood in the stool but negative barium enema and upper gastrointestinal series by x-ray?

DR. ENSBERG: I would suggest an air contrast barium enema study. Colonoscopy is an excellent study but it is time consuming and a difficult procedure for the patient and the colonoscopist.

DR. B. W. LARSON:‡ I would like to note that as you follow a patient's hemoglobin in the office, if the patient has a significant fall in the hemoglobin over a period of time, even though the value does not drop below the lower limits of normal, this patient should be further investigated. A common cause is occult gastrointestinal bleeding due to carcinoma of the gastrointestinal tract.

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* * *

Lonnie Waltner, M.D., Bridgewater, was named recipient of the Edwin J. Batt Memorial Award by the USD School of Medicine. Dr. Loren Amundson, Sioux Falls, presented the award on behalf of the Department of Family Practice. Dr. Waltner has practiced in the Bridgewater-Freeman area since 1968.

* * *

Dr. E. H. Collins was crowned King of the Gettysburg Centennial celebration. Dr. Collins, who began his practice in Gettysburg in 1935, has been very involved in the growth and development of that community.

* * *

R. C. Jahraus, M.D., Pierre, and David M. Patterson, M.D., Redfield, have completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

* * *

Gregg Tobin, M.D. has opened his general surgery practice in Winner. Dr. Tobin is a native of Winner. He graduated from the USD Medical School in 1978 and did his surgery residency at Sacred Heart Hospital, Yankton, and the VA Hospital in Sioux Falls. Dr. Tobin and his wife have two children.

* * *

Yankton's 1983 Citizen of the Year award was given to T. H. Sattler, M.D. Dr. Sattler has been practicing in Yankton since 1948. Dr. Sattler's interests in Yankton have not been confined to the field of medicine. He has served on boards of trustees for Yankton College, Mount Marty College, Yankton Carnegie Library, United Church of Christ, S.D. Board of Higher Educational Facilities. He currently serves on the Board of Trustees at Sacred Heart Hospital.

Jones County residents recently honored **Dr. Robert Hayes**, Wall, for his dedicated service to the community.

* * :

Robert Harner, M.D. has opened his practice in Custer. He is a general practitioner and a board certified surgeon. Dr. Harner came to Custer from Redding, Calif. He received his medical degree from Hahnamann Medical College in Philadelphia in 1951. He interned at the Los Angeles County Hospital and did a residency at the Merced County Hospital in Merced, Calif. He had a general practice in Atwater, Calif. for ten years. Dr. Harner and his wife have five children. He likes hunting and fishing.

* * :

Winston Odland, M.D. has been appointed as Vice-President of Medical Affairs at Dakota Midland Hospital in Aberdeen. Dr. Odland has practiced in Aberdeen for 14 years and is a past president of the South Dakota State Medical Association.

* * *

Recently elected to serve another term as Chairman of the Pathology Committee of the North Central Cancer Treatment Group, was John F. Barlow, M.D., of Sioux Falls. NCCTG is a cooperative cancer group of small cities over a several state area in the Upper Midwest which is associated with the Mayo Clinic and is dedicated to providing state-of-the-art treatment of cancer to patients in this region.

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S Future Meetings

October

- Update in Cardiology, Colony Square Hotel, Atlanta, GA, Oct. 10-13. Contact: ACC, 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.
- Principles of Colon and Rectal Surgery, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, Oct. 12-15. Fee: \$350. 26 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.
- 7th Annual Midwest Conference on Health Care in the Elderly, U. of Iowa Hosp. & Clin., Iowa City, IA, Oct. 14-15. 10.5 hrs. Category I credits. Contact: CME, U. of Iowa Coll. of Med., 285 ML, Iowa City, IA 52242. Phone: (319) 353-5763.
- CME Goals, Objectives and Evaluation: The Critical Link, Drake Oakbrook Hotel, Oak Brook, IL, Oct. 14-15. Contact: Illinois Council/CME, 55 E. Monroe, #3510, Chicago, IL. Phone: (312) 236-6110.
- Eighth Annual Perinatal Conference, Ramada Inn, Sioux Falls, SD, Oct. 17-18. 13 hrs. eredit applied for. Sponsored by SD Perinatal Assoc. and USD School of Med. Contact: Margo Varcoe, RN, SD Perinatal Assoc., 1100 S. Euelid Ave., P.O. Box 5039, Sioux Falls, SD 57117-5039. Phone (605) 333-7193.
- Emergency Medicine for Primary Care Physicians, St. Paul Hotel, St. Paul, MN, Oct. 19-21. Contact: Ruth K. McIntyre, CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Medical Determinations in Worker's Compensation, Ambassador West Hotel, Chicago, IL, Oct. 24-25. Contact: Am. Society of Law & Medicine, 765 Commonwealth Ave., Boston, MA 02215. Phone: (617) 262-4990.

November

- Primary Care of the Child with a Development Disorder, Gillette Children's Hosp., St. Paul, MN, Nov. 4-5. CME eredits. Contact: Gillette Children's Hosp., 200 E. Univ. Ave., St. Paul, MN 55101. Phone: (612) 291-2848.
- Implications of DRG Reimbursement for Hospital-Physician Relations, Howard Johnson's Motor Lodge, Rapid City, SD, Nov. 8. Contact: Gloria Roseland. Phone: 343-8550.
- 77th Annual Scientific Assembly, Hyatt Regency, Baltimore, MD, Nov. 6-9. Contact: Jeanette Stone, Southern Med. Assoc., P.O. Box 2446, Birmingham, AL 35201. Phone: (205) 323-4400.
- Implications of DRG Reimbursement for Hospital-Physician Relations, Sheraton Inn, Aberdeen, SD, Nov. 9. Contact: Kathy Graham. Phone: 229-4040.

- Newer Perspective in Human Lymphoma, Shamrock Hilton Hotel, Houston, TX, Nov. 9-12. Contact: Off. of Conference Services, Box 18, M.D. Anderson Hosp. & Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.
- Implications of DRG Reimbursement for Hospital-Physician Relations, Holiday Inn, Mitchell, SD, Nov. 10. Contact: Sue Wermers. Phone: 996-6501.
- Strategies and Controversies in Primary Care Medicine, St. Paul Hotel, St. Paul, MN, Nov. 10-12. Contact: Ruth K. McIntyre, CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Medical Complications of Pregnancy, Palmer House, Chieago, IL, Nov. 11-12. Fee: \$195. 14 hrs. Category I credits. Contact: Glory Ervin, Medical Complications of Preg., Dept. of OB/Gyn, Mount Sinai Hosp., 1500 S. Fairfield Ave., Chieago, IL 60608. Phone: (312) 542-2005.
- NIH Consensus Development Conference on Drugs and Insomnia, Masur Aud., Nat'l. Instit. of Health, Bethesda, MD, Nov. 15-17. Contact: Michele Dillon, Prospect Asso., Ste. #401, 2115 E. Jefferson St., Rockville, MD 20852. Phone: (301) 468-6555.
- Family Violence in the Deaf Community, Sheraton-Midway, St. Paul, MN, Nov. 30-Dec. 1. Contact: Ruth K. MeIntyre, CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Sonography Course in Obstetrics and Gynecology, Westin Hotel & Mount Sinai Hosp., Chicago, IL, Nov. 30-Dec. 2. Fee: \$145. 7 hrs. Category I credits. Contact: Glory Ervin, Sonography Course, Dept. of OB/Gyn, Mount Sinai Hosp., 1500 S. Fairfield Ave., Chicago, IL 60608. Phone: (312) 542-2005.
- Ericksonian Approaches to Hypnosis and Psychotherapy, Phoenix Civic Plaza Convention Ctr., Phoenix, AZ, Nov. 30-Dee. 4. CME eredit hrs. Contact: Jeff K. Zeig, M.D., The Milton H. Erickson Found., Inc., 3606 N. 24th St., Phoenix, AZ 85016. Phone: (602) 956-6196.

December

- Pediatrics Postgraduate Conference, Iowa City, IA, Dec. 1-3. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Advanced Cardiac Life Support, Iowa City, IA, Dec. 2-4. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Cardiology Today, Iowa City, IA, Dec. 6-9. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Coronary Heart Disease, Sheraton Midway Hotel, St. Paul, MN, Dec. 7-10. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.





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OUTH DAKOTA JOURNAL OF Medicine

Ear Disease and Hearing Loss, Pierre, South Dakota, 1962-1982

Clinicopathological Conference Thirty Year Old Caucasian Female With **Abdominal Pain of 11 Days Duration**

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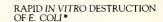
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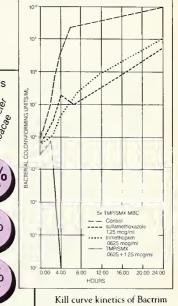
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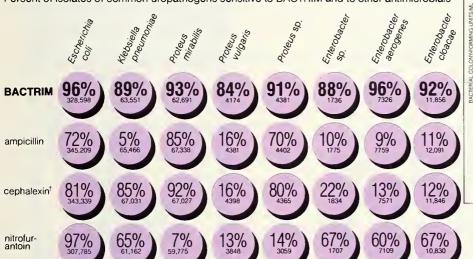
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Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.
Numbers under percentages refer to the projected number of isolates tested.

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*In vitro data do not necessarily predict clinical results.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, N.J. 2. Kramer M.J., Mauriz Y.R., Robertson T.L., Timmes M.D.: Morphological studies on the effect of subinhibitory and inhibitory doses of sulfamethoxazole-trimethoprim combination on Escherichia coli. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. 3. Spicehandler J et al. Rev Infect Dis 4:562-565, Mar-Apr 1982. 4. Stamey TA: Pathogenesis and Teatment of Urinary Tract Infections. Baltimore, Williams & Wilkins, 1980, p. 13. 5. Ronald AR: Clin Ther 3:176-189, Mar 1980. 6. Cooper J, Brumfilt W, Hamilton-Miller JMT: J Antimicrob Chemother 6:231-239, 1980. 7. Gower PE, Tasker PRW: Br. Med J 1:684-686, Mar 20, 1976. 8. Cosgrove MD, Morrow JW: J Urol 111:670-672, May 1974. 9. Iravani A et al. Antimicrob Agents Chemother 19:598-604, Apr 1981. 10. Schaeffer AJ, Flynn S, Jones J: J Urol 125:825-827, Jun 1981. 11. Rous SN: J Urol 125:228-229, Feb 1981. 12. BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82.

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For acute exacerbations of chronic bronchitis in adults due to susceptible For acute exacerbations of chronic bronchitis in adults due to susceptible strains of Haemophilus influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over a single antimicrobial agent. For enteritis due to susceptible strains of Shigella flexneri and Shigella sonnei when antibacterial therapy is indicated.

Also for the treatment of documented Pneumocysfis carinii pneumonitis.

Also for the treatment of documented Pneumocystis carinii pneumonitis. Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failuire when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

noted.
Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarm; reassess coagulation time when administering Bactrim to these patients.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

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Adverse Reactions: Alí major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasias: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Allergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. Gastrointestimal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colista and pancreatitis. CNS reactions: Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatique, muscle weakness and nervousness. Miscellaneous reactions: Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goltrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of golter pro-

certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies. Dosage: Not recommended for infants less than two months of age. URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN. AND ACUTE OTITIS MEDIA IN CHILDREN. Adults: Usual adult dosage for unirary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis. Children: Recommended dosage for children with uninary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis. For patients with renal impairment: Use recommended dosage regimen when creatine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is

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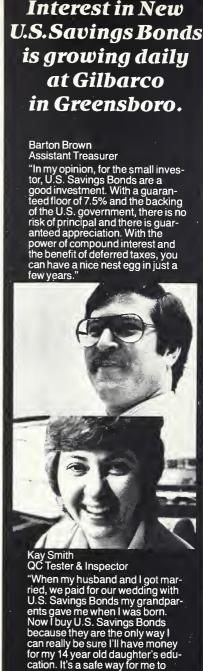
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24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

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Clinicopathological Conference Fifty-Six Year Old Caucasian Female With Infection of Knee and Acute Renal Failure

S Letters To The Editor

Dear Bob:

Thank you and the State Association for your kind remembrance of my 50 years of medical practice in Sioux Falls.

The plaque is truly beautiful, and I am proud to wear the pin you gave me.

Please convey my thanks to the membership.

Very truly yours,

Dr. Geoffrey I. W. Cottam

To the Members of SDSMA:

I am very grateful to have received the \$500.00 scholarship awarded by the S.D. State Medical Endowment Association, aiding me in my education at the University of S.D. Medical School in Vermillion, S.D.

Your generosity helps me obtain the many medical texts and journals that are needed by today's medical students for the future of tomorrow's doctors.

Thank you very much.

With deep appreciation, Marlys L. Schulz-Luebke

To the Members of SDSMA:

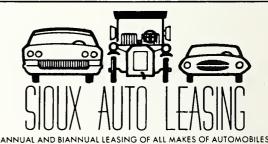
Thank you very much for the scholarship money you have donated and I have been awarded. It will be quite helpful in meeting this year's school expenses.

I will be starting my third year in June and am based in Sioux Falls. My career plans are to go where the Lord leads me, which may be to a mission field in another country to spread the good news of Jesus Christ and to help provide medical care to those in need.

Again, thank you very much for your generosity and the Lord bless you richly.

Sincerely, Paul J. Olson





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S Medicine

Ear Disease and Hearing Loss, Pierre, South Dakota, 1962-1982

John B. Gregg, M.D.* Kim Meyer Roberts, B.A.† Michael J. Colleran, M.A., C.C.C. (Audiology)‡

ABSTRACT

Disturbed hearing is the commonest disabling disorder in the U.S. today. A significant proportion of hearing losses begin during childhood and are related to infectious diseases, otitis media being the largest single cause. Clinical studies performed throughout South Dakota and at the Pierre Indian School in the early 1960s documented the high frequency and the severity of sequellae of otitis media, especially in the Native American population. Subsequent to these studies much effort and money was directed toward the treatment and prevention of this disease. To assess the efficacy of measures employed to control and prevent otitis media, in 1982 220 Pierre

Between 1955-1962 otology field clinics were conducted at Indian Reservations and at other places in South Dakota. Beginning in 1961 many special large population evaluations for ear diseases, hearing disorders, and speech pathology were performed on school and pre-school children, by a team representing the School of Medicine and the Speech and Hearing Clinic of the University of South Dakota. These voluntary, non-funded programs were undertaken because it had been found during the previous work that otitis media was the commonest infectious

pre-school children underwent clinical evaluations similar to those performed in the 1960s. Although the data compiled differ in many respects, a comparison of the two studies is possible and constitutes the substance of this report. Cautious extrapolation between the studies suggests that although otitis media is still prevalent in this region, there is less structural damage to the sound conduction mechanism today. In addition, asymptomatic otitis media occurred in 10% of the children evaluated in 1982. This suggests that otitis media must still be suspected in fussy children, those with unexplained fevers, and whenever there are unexplained gastrointestinal symptoms

disease in this region, especially in the Native American children, (Figures 1 & 2). Residua of otitis media and its most prominent functional complication, hearing loss, were very common. 1, 2, 3, 4 Often children with hearing losses did poorly in school but were designated as lazy, "backward" or retarded, because disturbed hearing was unrecognized. An additional factor contributing to the complications from otitis media was the cultural concept prevalent to many Native American families that a draining ear is merely a part of growing up. 5 The clinical studies by the University of South Dakota group were programmed to find the magnitude of the problem.

Before the mid-1960s there was no comprehensive program in South Dakota to screen school children for ear disease and hearing losses. It was not possible to provide routine otological examinations

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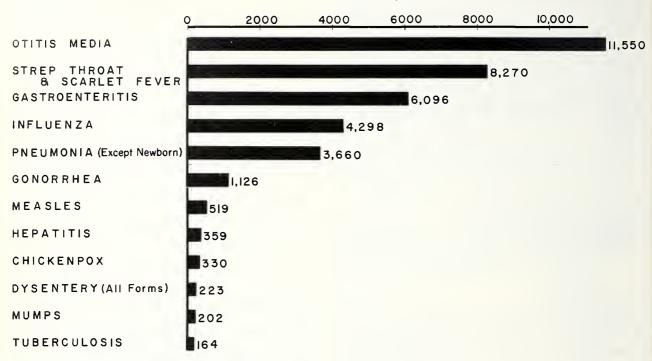
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INCIDENCE OF NOTIFIABLE DISEASES

INDIAN ABERDEEN AREA

REA INCIDENCE RATES

RATE PER 100,000 POPULATION



CALENDAR YEAR 1969

Figure 1
Incidence of notifiable diseases, Aberdeen Indian area, calendar year 1969, rate per 100,000 population. FROM: CHARTS on Indian Health, Aberdeen Area, Ed. 5. U.S. Dept. H.E.W., P.H.S., HSMHA, Indian Health Service.

for all children in the state on entry into the school system, or if hearing loss was suspected later. But it was feasible to evaluate each pupil's hearing audiometrically when he/she entered school, and thereafter if hearing impairment was suspected. If audiological testing was to be the only screening procedure utilized, in addition to establishing protocol to locate hearing impairments, it was necessary to investigate an promulgate measures to minimize false positive audiometric reports which lead to overreferral of children to definitive therapy from a test unit. Before making any hearing evaluation program operational, the logistics of such program had to be investigated systematically, so variables could be discovered and the mechanics of the project organized.

To assess the validity of audiometric testing alone as a means to detect clinically significant ear disease, during 1963-64, 385 children in grades K through 12 at the Pierre Indian School were evaluated by a multidisciplinary team, (Table I). An otolaryngologist examined all children and took a

brief history. If parents were present (very infrequently), they were interviewed. Each child was evaluated for conditions which could produce or accentuate a hearing loss, including active or previous ear disease, nasal and throat pathology, upper respiratory infections, outer ear canal obstruction by cerumen, adenotonsillar hypertrophy or acute infection, and congenital anomalies in the cranio-facial region. Because treatment was involved, ear canal obstruction by cerumen was recorded, but not removed. When hearing was abnormal, cerumen was removed and audiograms were redone.

Every child's hearing was tested in a special quiet room at the school with pure tone screening audiograms at the 10 decibel level (500, 1000, 2000 and 6000 c/s) and 20 db (4000 c/s) ASA 1951, following which each child's speech was evaluated seeking abnormalities indicating impaired hearing during the speech development period in life. To avoid any influence upon members of the different disciplines decisions as to the presence or degree of abnormalities the otolaryngologist, the audiologist, and the

REPORTED INCIDENCE SELECTED NOTIFIABLE DISEASES

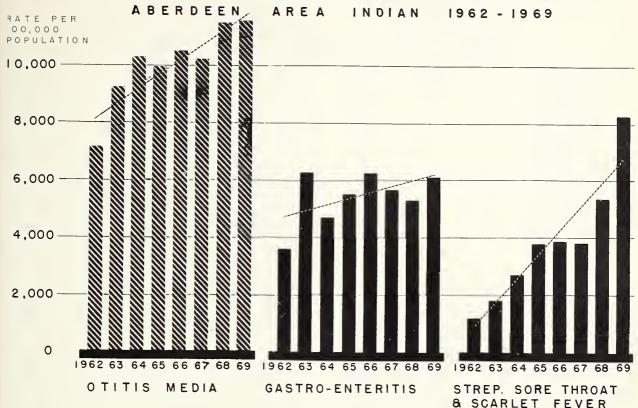


Figure 2
Reported Incidence, Selected Notifiable Diseases, Aberdeen Area Indian, 1962-1969, rate per 100,000 population. NOTE: increase in frequency of otitis media. This probably represents better reporting following demographic studies on various Dakota territory reservations, performed by team from the University of South Dakota. FROM: CHARTS on Indian Health, Aberdeen Area, Ed. 5. U.S. Dept. H.E.W., P.H.S., HSMHA, Indian Health service.

Pierre Indian S Radiographi		Distri						
		10-12 years			Total			
Radiograph study	group			_				
Male	19	20	32	_	71			
Female	19	35	22	1	77			
Control group								
Male	17	36	57	9	119			
Female	20	40	54	4	118			
	75	131	165	14	385			
Adapted from: Gregg et al 1970.								

speech pathologist made their determinations independently and the others' findings were not available between and among the team members at the time each child was being evaluated. At the conclusion of each test day, reports from the different disciplines were compared to find and evaluate the significance of discrepancies in the results.

Then, bilateral mastoid radiographs were taken of every other child at the school beginning in the first

grade, for a total of 151 individuals (TEST GROUP). The 242 children in the school upon whom mastoid radiographs were not taken formed the CONTROL GROUP. The radiographs were interpreted separately and collectively by two radiologists and one otolaryngologist, according to the precepts of Tremble. No clinical data relating to the children were available to the examiners during interpretation of radiographs.

Table II contains the clinical findings from this study. During the total project it was found that active ear disease (suppurative otitis media, serous otitis media, dry drum perforations) was missed audiometrically in 3 individuals (0.8%) from all the children tested. Residual changes from previous, probably infectious ear disease in a total of 130 ears (16.9%), manifested by clinical findings in 117 ears (15.2%) and radiographs of 13 ears (1.7%) showed no hearing disturbance which might impair the effectiveness of the individuals in school. The otolaryngic and radiographic results indicated that at least 24% of the test group had had significant ear

TABLE II
Pierre Indian School Ear-Hearing Survey, 1963-1965
Abnormalities found, ears and ENT area (tabulated by individual ears)

	Test Group (N = 153)				Control Group $(N = 242)$				$ \begin{array}{r} \text{Totals} \\ (N = 385) \end{array} $			
	Mild to Moderate		Severe	%	Mild to Moderate		Severe	%	Mild to Moderate		Severe	%
Ears		163		21.0		211		25.0		25.4		40.6
Clinically normal Active Processes		163		21.0		211		27.0		374		49.0
Supp. otitis media	1		7	3.0			1	0.2	1		8	1.0
Serous OM	4		í	2.0				0.4	6		1	1.0
Drum perforation	ī			0.3	ī		1	0.4	2		1	0
Cerumen obstructing	•			0.5	•		•	0.4	-		•	0
external canal	37		33	23.0	74		12	18.0	111		45	20.0
Residual processes								2010				
Scarring	30		9	13.0	46		2	10.0	76		11	11.0
Calcereous plaques	7		_	0.2	8		_	2.0	2		13	2.
Monomeric												
membrane	2			0.7	9		1	2.0	11		1	2.
Retraction of drum	17			6.0	30		1	6.0	47		3	6.
OTHER Otolaryngolic												
Findings												
NOSE												
Septal abnormalities,												
spurs, thickening,												
warping		63		41.2		84		34.7		147		38.
URI		40		26.1		27		11.2		67		17.
No abnormality		57		37.3		140		57.8		197		51.
ГHROAT												
Enlarged tonsils												
& adenoids		82		53.6		67		27.7		149		38.
Subacute tonsillitis		1		0.6		_						0.
Cleft palate, repaired		1		0.6		1		0.4		2		0.
OTHER												
Endaural surgery,						,		1.		4		
unilateral				_		4		1.6		4		1.
Endaural surgery, bilateral		1		0.6		1		0.4		2		0.
Facial paralysis,		1		0.6		1		0.4		2		U.
unilateral						1		0.4		1		0.

disease in the past, but this caused hearing impairment in only a small fraction of the examinees. A significant truism which emerged from the study was that the past medical history provided by the Indian School children was a poor method to determine the likelihood of previous ear disease or affectation of the hearing mechanism.

Two conclusions emanating from this study were, 1) audiometric studies done as described are reliable to detect middle ear pathology which present as hearing loss, and as a means for screening large school population groups, and 2) over-referral of children from hearing screening programs is a constant problem, but can be minimized through appropriate policies for referral of those with sensori-neural loss, and retesting of those found with mild conductive losses, prior to medical consultation.

The University of South Dakota hearing evaluation programs stimulated much interest concerning ear diseases and hearing problems throughout the state, especially in school aged children. Over a 10 year interval this concern was manifested in several ways, which included,

- 1) the Easter Seal Society awarded a grant to the Speech and Hearing Clinic of the University of South Dakota to construct and maintain a mobile hearing testing laboratory which served the entire state.
- 2) the training programs for audiologists and speech pathologists at the University of South Dakota received increased academic and financial support. As a result, many individuals were trained in these fields, and now the services of speech pathologists and audiologists are available to most South Dakota communities.
- 3) when the U.S.P.H.S. Indian Health Service became aware of the findings from the University of South Dakota WET BONES otitis media project, increased effort and funding were channeled into the control of this widely prevalent and potentially very serious disease.

- 4) the University of Nebraska Otolaryngology Department received a large three year grant for a project to control otolaryngic diseases, especially otitis media, on the Pine Ridge Reservation (the Pine Ridge Reservation was identified by the University of South Dakota WET BONES survey as having the greatest number and the most severe residua of otitis media).
- 5) beginning about 1877 and still operational, the Indian Health Service employed a Physician's Assistant specially trained in recognition of otological problems, to serve the Indian reservations in this region, screening for ear disease and referring problems found to sources of definitive therapy.

6) during the interval 1978-1980, the Visiting Professor representing jointly the School of Medicine, USD, and the State Health Department traveled throughout South Dakota on a regular basis, emphasizing otitis media (diagnosis, treatment, prevention), and continuing health education.

7) educational programs directed toward the causes of ear diseases and their prevention, aimed at health care personnel, teachers, and the public, have been conducted throughout the state during the past 15 years.

After almost 20 years work on the problem of otitis media in South Dakota, it was advantageous to determine whether any recognizable improvement had taken place in the epidemiology of this disease. The opportunity to do this became available in August 1982, as part of the pre-school screening of a large group of children at Pierre, SD. Although not totally comparable, the results from the 1982 evaluations allow some insight into otitis media, in mid-South Dakota, over a 20 year interval. A comparison of the results in 1970,⁶ and the clinical findings of today constitute the substance of this report. The 1982 audiological findings will be reported later.

MATERIALS AND METHODS

In mid-August 1982, 220 children aged 5.0 to 6.5 years, underwent preschool examinations provided by the Pierre Public School system. Otolaryngic examinations, hearing evaluations, and assessment by a speech pathologist, were included in the studies. As a corollary to the audiometric studies, and to assay the efficacy and cost effectiveness of different methods to test auditory function, the childrens' ears were evaluated with three different testing modalities. These included, 1) pure tone audiograms, 2) tympanometric evaluations, and 3) V A S C (Verbal Auditory Screening Test for Children, a modified method of speech audiometry adapted for mass screening).

The parent who accompanied each child to the test site supplied written information relating to past

health on a short medical history form by answering these questions:

- 1. Has your child had earaches? _____ If so, how often? _____
- 2. Have you seen any discharge or drainage from your child's ears?
- 3. Has your child received medical treatment for any ear problem? _____
- 4. Does your child have a known hearing loss?

5.	Has yo	ur child	experienced	tinnitus	(ringing	in
	the ears	s)?				
	If so:	Right e	a r	Left ear	r	

6. Has your child been exposed to excessive loud

Optional (You may or may not choose to complete this portion)

7.	Your family's total yearly income: (check one)
	0-\$2,000 10,000-19,999
	2,000-4,999 20,000-29,999
	5,000-9,999 30,000-over

Question #7 was important to locate any correlation between socio-economic factors and clinical evidence of ear disease or hearing loss.

Insofar as possible, technically the 1982 evaluations paralleled those of the 1960s, but no radiographic studies were proposed for 1982. Variables and commonalities between the 1960s and the 1982 study included the following:

- 1. The children examined in 1982 were all between 5.0 and 6.5 years of age. The Indian School children ranged from 6-18 years. To compare findings from the two studies, the 1960s Pierre Indian School data were recompiled to reflect the age groups 6-7 yr., and 8-9 yr.
- 2. Most of the Indian School children were examined in late October and had had greater opportunity for exposure to upper respiratory infections before or at the time of testing. The 1982 study was done in mid-August so there was less likelihood of the effect of respiratory infections upon the middle ears
- 3. The Indian School pupils were ethnically homogeneous, while six ethnic groups were represented at the Pierre Public School (Caucasian N = 202, Native American N = 14, and one each India, Japanese/American, Korea, and Oriental). The small number of Native American children in the 1982 study limited the statistical information which could be extrapolated. Unfortunately, no data from a comparable 1960s non-Indian School population were available for purposes of comparison.
- 4. Socio-economic conditions and educational opportunities were quite different between the 1960s and 1982 populations.

TABLE III
Pierre Indian School Hearing Study, 1963-1965
Results for children ages 6-9 years.

	Age 6-7 yr. $(N = 29)$		Age 8-9 yr. $(N = 46)$			Total $(N = 75)$			
	Individuals	%		Individuals	%		Individuals	%	\
HISTORY of Ear Disease or Hearing Loss (available for 38 individuals)	4/17	23.5		6/21	28.6		10/38	26.3	
EXAMINATIONS	Individuals	%	\mathbf{R}/\mathbf{L}	Individuals	%	R/L	Individuals	%	R/L
EARS									
Scarring	2	6.9	4/4	4	8.7	8/8	6	8.0	12/12
Retraction	4	13.8	4/4	6	13.0	5/4	10	13.3	9/9
Monomeric membrane	1	3.4	-/1	_	_	-/-	1	1.3	-/1
Perforation		_	_	3	6.5	-/3	3	4.0	-/3
Serous OM	2	6.9	2/1	_		_	2	2.7	2/1
Discharge	-	_	_	41	8.7	-/4	4	5.3	-/4
Cerumen obstructing external canal	4	13.8	4/4	18	39.1	14/10	22	29.3	18/14
NOSE & THROAT									
Nasal septal abnormality	7	24.1		13	28.3		20	26.6	
Enlarged or infected tonsils	18	64.3		23	50.0		41	54.6	
Cleft palate	1	3.4			_		1	1.3	
Bifid uvula	1	3.4			_		1	1.3	
URI	11	38.0		10	21.7		21	28.0	
HEARING LOSS	11	38.0	8/8	14	30.4	13/14	25	33.3	2:1/22
RADIOGRAPHS abnormal (available									
for 38 individuals, 76 ears)	4/17	23.5	2/4	8/21	38.1	5/4	12/38	31.6	7/8

T/ABLE IV
Pierre Public School Otologic and Hearing Study, 1982
(Historical and Otologic Aspects)
Results for 220 preschool children ages 5.0-6.5 years.

	Genera population			Native A		ans	Othe India		origin (one Jap./Amr	
MICHODA	Individuals	%	R/L	Individuals	%	R/L	R/L	R/L	R/L	R:/L
HISTORY										
Earaches	99	49.0		4	28.6	_	_	1	1	
Discharge	28	13.9		1	7.1	_	_	_	_	_
Treatment	88	43.6		2	14.3		_	_	_	
Hearing loss	8	4.0		_	_	_	_	_	_	
Tinnitus	2	9.9		_	_	_	_	_	_	
Noise exposure	9	4.5		_	_	_	_	_	_	_
EXAMINATION										
EARS										
Scarring or translucency	59	28.7	54/42	5	35.7	5/3	-/1	-/1	_	
Retraction	14	7.0	13/10	_	_	_	_	_	_	
Monomeric membrane	2	0.9	-/2	_	_			_	_	
Perforation	_	_	_	_	_	_	_	_	_	
Serous OM	2	0.9	2/1	_	_	_	_	_	_	_
Discharge	_	_		_		_	_		_	_
Bulging	1	0.5	-/1	_		_	_	_	_	
Cerumen obst. ext. canal	11		11/9	_		_	_		_	_
NOSE & THROAT		0.4	11//							
Nasal septal abnormality	31	15.3		4	28.6	_	_		1	1
Enlarged or inf. tonsils			te enla	argement in	all ch	ildren ections.		remove	d from 4.	No acute
Cleft lip or palate	1	1.5		_		ctions			_	_
Tedinous raphe	î	1.5		_	_				_	
Bifid uvula 2-3+	î	1.5								
URI	25	12.4		1	$\frac{-}{7.1}$					

- 5. The Pierre Public School children lived at home while the Indian School children were living in school dormitories.
- 6. The ratio of males to females was approximately equal in both study groups. No sex preponderance for ear disease or hearing problems was found, so the data are not compiled by sex. However, because ear disease and hearing loss were found unilaterally and bilaterally, data are arranged to reflect the individuals involved and the number of ears affected.
- 7. The children in both study groups had access to health care, but the opportunity for treatment of diseases early in life was limited for many Indian School children.
- 8. The factor common to all children in both studies was that mostly they had been born into and all were living in the same geographic area.

RESULTS AND CONCLUSIONS

The findings obtained in children aged 6-9 years during the study at the Pierre Indian School during the 1960s, are in Table III. The results at the Pierre Public School in 1982 are in Table IV. To facilitate comparison between the two studies, the 1982 data have been subdivided according to ethnic groups. Although the 1982 Native American sample size is small, it is possible to discern patterns which can be compared to the findings at the Indian School in the 1960s.

The most important information derived from these tables is that otitis media is still very prevalent in this region, but serious structural side effects accuring from it are minimal and appear to be decreasing in number and severity. Evidence of acute and chronic mastoiditis as in days of yore, was not reported in the 1982 study. Important considerations emanating from this study include the following:

1. Medical Historical Information.

A. Whereas information obtained from the Indian School children in the 1960s indicated that there had been bouts of otitis media in about 25% of the 6-9 year old children, 49% of the Pierre general school population and 28.6% of the Native Americans had histories of previous middle ear disease. This is interpreted to represent better reporting rather than increased frequency of otitis media.

B. In the Pierre Public School population, 22 mothers (10%) reported no earaches in their children, but that there had been aural discharge and/or treatment for infectious ear disease.

C. Thirty (14.8%) Public School general population and one Native American (7.1%) mothers acknowledged previous earaches or aural discharge but reported no medical treatment for the condition. In 11/30 of these children clincial evaluation revealed

evidence of previous middle ear disease which was of mild to moderate severity in 10 and pronounced changes in one child. Mild residual of otitis media was bilateral in six youngsters and unilateral in 4, while the pronounced changes were bilateral. In 2/30 children the external auditory canals were obstructed by cerumen such that clinical evaluation was not possible.

D. In reply to the question regarding family income, these answers were forthcoming: 0-\$2,000. N = 2; 2,000.-4,999. N = 3; 5,000.-9,999. N = 10;10,000.-19,999. N = 41; 20,000.-29,999. N = 41;30,000.-over N = 35; No response N = 73. Because 73/220 mothers (33.2%) had not indicated the family's income level, it was not possible to obtain as much information as was desired to compare ear problems with income levels. Of the 15 Public School children in the three low level income groups, seven had findings suggesting residua of otitis media, all of mild to moderate severity, bilateral in six. Two of these children with positive physical findings had received medical treatment, and one with historical but not clinical otitis media had been treated. In two other children with one normal ear, the other ear could not be inspected due to external canal obstruction.

2. Clinical Findings, EARS.

A. Scarring and translucency of the tympanic membranes was recorded in 8% of the Pierre Indian School children, in 28.7% of the Public School general population, and 35.7% of the Public School Native Americans. For the most part this was of only mild to moderate degree. The apparent increase in frequency is interpreted as being more the result of critical appraisal of physical findings than as a significant increase in numbers of affected ears.

B. Drum retraction was found in 7% of the Public School pupils, in 13.3% of the Indian School pupils, and in none of the Public School Native Americans. This difference is considered significant and probably reflects the effect of upper respiratory infections upon the middle ear by virtue of differing climatic conditions at the time of the two studies.

C. Monomeric membranes were present in 0.9% of the Pierre Public School children, 3.4% of the Indian School pupils, and in no Public School Native Americans. This finding suggests less effect of otitis media upon the tympanic membranes of the children tested in 1982.

D. No tympanic membrane perforations were found in Public School children, but were present in 4% of the ears of the Indian School children. This finding is considered definitely significant, indicating lesser effect of otitis media upon the sound conduction mechanism in 1982.

E. Serous otitis media was identified in 0.9% of the Public School children (none in Native Americans or other ethnic children) but was found in 2.7% of the Indian School pupils. The greater numbers found in the 1960s could have been the result of climatic differences at the time of testing. Five Pierre Public School general population children and one Native American child had polyethylene (PE) tubes in their ears at the time of examination, indicating previous treatment for serous otitis media. None had been present in the 1960s. All the children with PE tubes showed residual changes in their tympanic membranes.

F. Aural discharge was found in 5.3% of the Indian School youngsters but was not present at the Public School. This difference indicates less effect of otitis media upon the drum and middle ears in 1982.

G. External auditory canal obstruction by cerumen was recorded for 5.4% of the total Public School group and was noted in 29.3% of the Indian School pupils. Although more ear canals were obstructed at the Indian School, followup studies showed that hypercerumenosis had not caused significant conductive type hearing impairment or did not obfuscate underlying middle ear disease (unpublished data).

3. Clinical Findings, NOSE & THROAT.

A. Nasal septal deflections, warping, thickening, and spurs, were found in 15.3% of the Public School general population, 28.6% of the Native Americans, and 26.6% of the Indian School children. The higher frequency of nasal septal abnormalities in Native Americans than in Caucasians mirrors findings in other studies performed by the University of South Dakota team (unpublished data).

B. As might be expected, the children in both study groups frequently had adenotonsillar hypertrophy. Only occasionally this could be correlated directly with residual changes of otitis media.

C. Congenital palatal clefting, tendinous palatal raphe, and definite bifid uvula, all forms of inborn non-odontogenic fissural defects, were found in 3/220 (1.4%) of the Public School population, and 2/75 (2.7%) of the Indian School pupils. The higher frequency of inborn anomalies in the palatal-facial region in the Native Americans parallels the findings by others in this region. ^{8, 9} Inborn palatal abnormalities are important in that there is a strong correlation between palatal dehiscence and repetitious and chronic middle ear disease, and conductive type hearing loss. The Public School child with lip and palate defects had had much middle ear disease and treatment for it (PE tubes and mastoid surgery), the child with cleft uvula had moderately severe residua

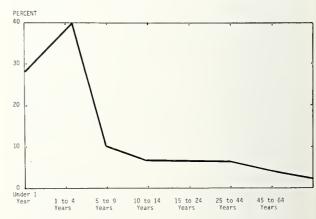
of otitis media but no history of ear infections or treatment, and the child with a palatal tendinous raphe had no history or physical findings indicating previous otitis media. The Indian School child who had undergone palatal repair had a history of previous ear infections. Examination showed serious otitis media on the right and ear canal obstruction on the left. Audiograms indicated moderately severe mixed type hearing loss on the right, and mastoid radiographs were interpreted to show severe bilateral alterations resulting from previous otitis media. The Indian School child with bifid uvula had no historical, physical, or radiographic evidence of previous middle ear disease.

D. URI were present in 12.4% of the Public School general population and 7.7% of the Native American children, and in 28% of the Indian School pupils. The greater number of infected noses found in the 1960s is best explained as secondary to the environmental variables between the two studies.

DISCUSSION

Hearing losses are the commonest disabling disorder in the United States today. They become manifest most frequently at the two ends of the spectrum of life, the very young and the elderly. A very high proportion of hearing losses which begin early in life (and often persist throughout life) are of infectious origin and are preventable. The frequency pattern by age groups, found by the Indian Health Service office during the evaluation of a large group of individuals is depicted in Figure 3. It is notable that the frequency of otitis media drops sharply after about the sixth birthday.

The findings from this study indicate that although otitis media is still very prevalent in mid-South Dakota, the attacks which are occurring in



Information source: Aberdeen Area, Indian Health Service Office, Aberdeen, 5.0.

Figure 3
Percent distribution of acute cases of otitis media by age, Indians and Alaska Natives, CY 1976. Information Source: Aberdeen Area, Indian Health Service Office, Aberdeen, SD

children in the age bracket under study are producing less structural change in the ear drums and middle ears. Some of this undoubtedly reflects different socioeconomic factors between the 1960s and the 1982 study groups. It appears that progress has been made in eliminating the ill effects of otitis media in mid-South Dakota during the past 20 years, if not the disease itself.

A significant finding which eminated from the 1982 investigation is the fact that 22/220 (10%) of the children had no history of earaches, but had required treatment for ear infections. In three of these children, aural discharge announced the presence of infectious ear disease. This indicates that asymptomatic otitis media must still be considered in the instance of a "fussy child," "obscure febrile symptoms," sleeplessness, and unexplained gastrointestinal symptoms, especially persistent diarrhea. Asymptomatic otitis media is more frequent in very young children. ¹⁰

An additional significant finding is the fact that 29/202 (14.4%) of the Public School white population and 1/14 (7.1%) Native American children had had infectious middle ear disease by history but had received no medical treatment. Of these 34.5% had clinical evidence of previous middle ear disease, albeit for the most part very mild. Because of many mothers' reluctance to declare family income, it was not possible to correlate this factor with socioeconomic conditions in the home.

The major thrust for the future must be continuing emphasis upon prevention of otitis media. This will be accomplished best through measures of hygiene, prompt treatment when this disease is found, and awareness of the fact that asymptomatic otitis media is still very prevalent in this region. Three most important facets in this program are, constant awareness of the prevalence of otitis media by members of the health care team, education of mothers and prospective parents concerning this problem, and appropriate screening of preschool and school children to detect evidence of middle ear pathology.

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102nd Annual Meeting of the South Dakota State Medical Association



House of Delegates in session.



Dr. Joseph Hamm was introduced as the 1983-84 president by outgoing president, Dr. Durward Lang, at the banquet held Saturday evening, June 4, at the Ramada Inn, in Sioux Falls.



Dr. A. J. Barrett presided as Speaker of the House of Delegates. Dr. Gerald Tracy (seated) served as parliamentarian.



Enjoying the cocktail party are Virginia Tracy, Constance Rial, Dr. William Rial, AMA president, and Dr. Gerald Tracy.



Kay Reaney, AMA-ERF chairman, presents a check for \$21,157.20 from AMA-ERF to Dr. Robert Quinn, Dean of USD School of Medicine, at the First House of Delegates meeting. Looking on are Dr. A. J. Barrett, Dr. Durward Lang and Bob Johnson.



Dr. Richard Porter and Dr. Frank Messner enjoying the evening.



Involved in conversation are Dr. Richard Renka, Dr. James Wunder and Peggy Wunder.

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South Dakota Journal of Medicine 608 West Avenue, North Sioux Falls, South Dakota 57104

19

S This Is Your Medical Association

Drs. A. P. Reding, Marion; **Alvin R. Scheffel,** Redfield; and **Theodore A. Angelos,** Canton, have completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

* * *

A Doctor Otey Day was held in Flandreau to honor **Bernard T. Otey, M.D.** who will be retiring after 30 years of service in that community.

* * *

Dr. Wesley D. Putnam, Sioux Falls, was recently informed of his successful completion of all requirements, including board examination, for special competency in hematopathology. The American Board of Pathology has certified Dr. Putnam as a subspecialist in Hematopathology.

* * *

Drs. D. G. Ortmeier and R. E. Van Demark, Sr., both of Sioux Falls were elected to the Blue Shield Board of Directors. Dr. Ortmeier was re-elected Chairman of the Board and Roscoe Dean, M.D., Wessington Springs, was elected Vice-Chairman.

* * *

Dr. Bernard Heilman, a native of Aberdeen, has joined the Huron Clinic in family practice medicine. After graduating from high school, Dr. Heilman went to the U.S. Navy Hospital Corps School in Great Lakes, Ill. and then to the USD School of Medicine for his medical degree. He completed a three-year family practice residency at St. Joseph Mercy Hospital in Mason City, Iowa before coming to Huron. Dr. Heilman and his wife Linda have three sons.

Dr. Edwin Gerrish has entered a general surgery practice in Watertown, at the Brown Clinic. Dr. Gerrish received his medical degree from the USD Medical School and did his residency at Case Western Reserve Univ. in Cleveland, Ohio. Dr. Gerrish's wife, Cathy, is board certified in internal medicine and will also practice at the Brown Clinic.

* * *

Anthony Silvagni, D.O. has joined a family practice group in Parkston. He was born in Atlantic City, NJ. He received a Doctor of Pharmacy degree in 1970 and received his Doctor of Osteopathic Medicine degree in 1982. He completed his residency at the Tuscon General Hospital in Tuscon, Ariz. Dr. and Mrs. Silvagni (Diana) have two children.

* * :

Governor Bill Janklow recently appointed **Dennis Johnson**, **M.D.**, Sioux Falls, to the State Board of Medical and Osteopathic Examiners.

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ENDOWMENT FUND

S President's Page



"Providing one day outpatient services in: facial plastic surgery, breast augmentation, hair transplantation!" This ad in a publication of Piedmont Airlines in November, 1982 was a real attention getter. To my generation of physicians such a sales promotion conjures up memories of itinerant medicine shows, nostrums and quackeries of the past. We will see more marketing techniques used by ethical practitioners. There will be increasing need to reassure the public that full medical licensure is one requisite to optimal health care and that medicine is a profession and not a trade.

Marketing is often mistakenly perceived to be synonymous with advertising and selling. For health care, advertising is the least important marketing technique. Investor-owned medical conglomerates operated for profit are the major pressure moving the medical practitioner into advertising and marketing. With extensive resources, they research, analyze, create services for, package for, and advertise to their markets.

Individual physicians can take heart, however. The magazine cited above reported a poll taken by a sales and marketing association to determine the reasons why businesses lost customers. The results were revealing: 68% quit because of indifferent attitudes on the parts of the providers and employees; 14% — product dissatisfaction; 9% — competitive reasons, and 9% — were influences by others, moved away or died. Physicians may lack resources and expertise to compete with the corporate merchandising of medicine, but we can have the advantage in retaining the 82% of our "customers" who prefer a caring attitude and quality care.

Joseph N. Hamm, M.D., President South Dakota State Medical Association

S Chapter News

R for the 80s





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

AMERICA'S FAMILY DOCTORS SET OCTOBER AS "FAMILY HEALTH MONTH"

Fall is the time of year when people go back to school, change jobs, move to another city, or prepare for the cold months ahead. It also is a time to pay particular attention to health.

The American Academy of Family Physicians (AAFP), the national association of family doctors, has designated October, the first full month of autumn, as "Family Health Month" to help people focus on their families' health.

All over the country family doctors are encouraging people to take a close look at their families' eating habits, physical fitness, mental health and possible hazards in their homes. Americans are being urged to establish a "partnership for health" with a family doctor because he or she can provide total health care on a continuing basis.

For example, family doctors are trained to help patient-families with proper nutrition, good exercise habits, and dealing with stress, as well as 85-90 percent of all biomedical health problems. They serve also as health advocates to the patient, referring to appropriate consulting specialists those 10-15 percent of problems they are not trained to handle, while maintaining close contact even after referral and reassuming full responsibility thereafter.

"Family physicians focus on preventive as well as curative medicine, and educate patients and families in ways to stay well and healthy," said Dr. Harmon E. Holverson, incoming AAFP president. "In today's strained economic situation, our kind of preventive medicine — keeping people well and out of sick beds — is very cost-effective. Comprehensive, continuing family health care is the best health value going."

The AAFP is the nation's second largest medical group, with more than 55,000 members. The AAFP, headquartered in Kansas City, was instrumental in establishing the medical specialty of family practice in 1969. It also is a pioneer in continuing medical education (CME), requiring its members to earn 150 hours of approved CME credit every three years.

KEMP NEW DIRECTOR

The Sioux Falls Family Practice Residency Program named Earl D. Kemp, M.D. as its third full time Director. Bruce Vogt, M.D. served as Acting Director from October 1982 until July 1, 1983.

Dr. Kemp is a graduate of the University of Iowa College of Medicine, and interned at McKennan Hospital in Sioux Falls. Following several years of family practice with the Central Plains Clinic in Sioux Falls, Dr. Kemp joined the SFFPRP faculty as an Associate Director July 1, 1981.

We wish him well!

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CME CREDIT: AAFP: 12 Hours Prescribed AMA: 12 Hours Cat. I

Registration Fee: \$100.00

Course Objective:

The primary objective is to provide SDAFP members and guest registrants a continuing exposure to a cyclic core of knowledge in family medicine.

Content Areas:

Musculoskeletal Disorders, Including Arthritis
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For Information Contact:

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knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

IMECTABLE To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used IV: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veius, i.c., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Prccautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, harbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopta, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity,

insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia. In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported. Dosage: Individualize for maximum beneficial effect.

ORAL Adults: Anxiety disorders, relief of symptoms of anxiety—Valium (diaze-pam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: <u>Tablets</u>—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). <u>Capsules</u>—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). <u>Capsules</u>—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.) For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or urist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly IV, it may be injected slowly through the infusion tubing as close as possible to the vein insertion.

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg l.M. or l.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg l.M. or l.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg l.M. or l.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg l.M. or l.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children administer l.V. slowly; for tetanus in infants over 30 days of age, 1 to 2 mg l.M. or l.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred.). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred.): repeat in 2 to 4 hours if needed. EEG monitoring may be helpful. In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets — 2 mg, white; 5 mg, yellow; 10 mg, blue — bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules — 15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.



S Clinicopathologic Conference

Thirty Year Old Caucasian Female With Abdominal Pain of 11 Days Duration

In honor of John F. Barlow, M.D. for publishing CPC's monthly for seventeen years.

Delwin K. Ohrt, M.D.* Robert Raszkowski, M.D.† Discussers

Richard A. Jaqua, M.D.‡ Guest Editor

Case #A70-17

In late January of 1970 a 30-year-old white female was transferred to Sioux Valley Hospital because of persistent right upper quadrant abdominal pain, nausea and vomiting of 11 days duration. Her symptoms had not been alleviated by prochlorperazine suppositories for nausea and pentazocine for pain. She was admitted with a provisional diagnosis of cholecystitis and cholelithiasis.

The patient had only seen her home physician on two previous occasions. Four months prior to this hospitalization she had experienced similar symptoms, accompanied by a low grade fever and morning diarrhea. An oral cholecystogram failed to visualize the gallbladder on two consecutive days and an upper gastrointestinal series revealed a 5mm antral ulcer. No jaundice was noted at this time and no bile was noted in the urine. The patient was treated with intravenous fluids, antibiotics and antacids and became asymptomatic.

Approximately four and a half years prior to her hospitalization, the patient was first seen by her home physician who noted apparent jaundice, bile in the urine, and clay colored stools which persisted for approximately one week. There was no fever or elevation of the white blood count at this time. Hypertension of 214 systolic and 110 diastolic was noted and the patient was begun on chlorthalidone and a low salt diet.

The patient had always been markedly obese. No other members of her family were noted to be obese and the hospital records did not note any significant familial illnesses.

PHYSICAL EXAMINATION: Grossly obese white female complaining of intermittent right upper quadrant abdominal pain. Blood pressure 165 systolic and 80 diastolic; pulse 180/min and regular; repirations 24/min; temperature 37° C oral; height 5'3"; weight 131 kgms. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The examination of the heart revealed tachycardia but was otherwise normal. "Fairly marked" right upper quadrant tenderness to abdominal palpation was noted. Obesity was felt to preclude the exclusion of organomegaly and to compromise the rectal and pelvic examination. The remainder of the physician examination was within normal limits.

HOSPITAL COURSE: The patient was treated conservatively over the first two days of her hospitalization with a liquid diet, intravenous fluids and pain medication. The right upper quadrant pain persisted. Initial laboratory studies revealed: hemoglobin 11.7 gm/dl, hematocrit 36 vol/dl, normal red cell indices, total leukocyte count 10,000/mm3 ($10.0 \times 10^9/\text{L}$) with 71% segmented neutrophils, 0% neutrophilic bands, 1% eosinophils, 0% basophils, 26% lymphocytes and 2% monocytes. The sedimentation rate was 20 mm/hr. The total bilirubin was 0.4 mg/dl, blood urea nitrogen 26 mg/dl, and serum creatinine 1.1 mg/dl. The sodium was normal at 139 meq/L but the potassium and

Pathologist, Laboratory of Clinical Medicine, Mankato, MN, Clinical Assistant Professor of Laboratory Medicine, School of Medicine University of South Dakota, Sioux Falls, SD.

[†] Specialist in Gastroenterology, Sioux Valley Hospital, Associate Professor and Chief, Section of Gastroenterology Department of Internal Medicine, School of Medicine, University of South Dakota, Sioux Falls, SD.

[‡] Professor and Chairman, Department of Laboratory Medicine, School of Medicine University of South Dakota, Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.

chloride were depressed at 2.6 meq/L and 93 meq/L respectively. The urinalysis revealed a trace of proteinuria and innumerable red cells/hpf. The chest x-ray was normal. An intravenous cholangiogram revealed "a faintly opacified gallbladder which seems to be slightly larger than usual. The distal portions of the right and left hepatic ducts, common hepatic duct and common bile duct are not optimally opacified but can be discerned and there is no evidence of any filling defect of dilatation. There is a faint suggestion of some contrast medium in the duodenal loop."

On the third hospital day the patient began having melanotic stools and the hemoglobin dropped to $9.9~\rm gm/dl$. The potassium had risen to $2.9~\rm meq/L$ and the chloride to $95~\rm meq/L$. Two units of blood were administered and the vital signs remained stable. The next morning the hemoglobin was $11.4~\rm gm/dl$ but following episodes of hematemesis, the hemoglobin fell to $9.0~\rm gm/dl$. The pulse rose to $140/\rm min$ and the blood pressure was noted to be as high as $175~\rm systolic$ and $120~\rm diastolic$.

On the fifth hospital day the pulse rate was 200/min and the blood pressure was noted to be as low as 104 sytstolic and 70 diastolic. The patient was taken to the operating room where a large indurated area was identified in the region of the pylorus. A longitudinal incision across the pylorus revealed a 1.8 cm duodenal ulcer with a bleeding vessel at its base. The bleeding vessel was ligated and a vagotomy and pyloroplasty were performed. A large stone was found impacted in the lower end of the cystic duct and a cholecystectomy was performed.

On the first postoperative day the patient was described as significantly improved. However, that evening the temperature rose to 39.5°C, her sensorium clouded, and she was noted to have intermittent jerking movements of the extremities. Intermittent melena and hematochezia were noted. She was treated with blood replacement, three antibiotics, and both topical and systemic measures for fever.

Over the next three days the temperature fluctuated but was always elevated. The lungs remained clear and no definite infiltrates were noted on the chest x-ray. Arterial blood gases on O₂ by mask were: pH 7.52, pO₂ 85 torr, pCO₂ 48 torr, CO₂ content 39 mm/L, O₂ saturation 97%. The serum calcium was 9 mg/dl and potassium 3.1 meq/L. The hemoglobin as maintained at approximately 10 gm/dl with multiple blood transfusions.

Late in the evening of the fourth postoperative day the temperature rose to 41°C rectally. Early on the morning of the fifth postoperative the temperature remained 41°C despite attempts at external and internal cooling. Blood pressure was 116 systolic and 80 diastolic, pulse 200/min, and respirations 44/min. The pulse and blood pressure plummeted rapidly over a three hour period despite intravenous fluids and the patient expired early on the morning of the fifth postoperative day.

An autopsy was performed.

DR. OHRT: This young woman presented with signs and symptoms most consistent with gallbladder disease. Upper gastrointestinal hemorrhage became a major complication during the initial portion of hospitalization forcing operative intervention. She developed peritonitis following surgery and expired after a short, stormy course. This set of circumstances is sufficiently unusual to be recognized as a "sentinel event"; an event potentially important to our continuing medical education.

The autopsy findings are listed in Table I.

Our purpose for this gathering today is twofold. First, Dr. John Barlow, has published a CPC monthly for seventen years in the SOUTH DAKOTA

TABLE I

- I. Multiple endocrine adenomatosis
 - A. Islet cell adenomas of the pancreas
 - B. Hyperplasia of parathyroid glands
 C. Chronic duodenal ulcer with hemorrhage
 - (operated 2-3-70)
 - D. Breakdown of pyloroplasty suture line
 - E. Generalized peritonitis
 - F. Acute esophagitis, severe, with perforation into right pleural cavity
 - G. Right pleural effusion, (300 ml., estimated)
 - H. Hepatic congestion
 - I. Left pleural effusion (150 ml., estimated)
 - J. Bilateral lower lobe atelactasis
- II. Right pyohydronephrosis (approximately 70 grams)
- III. Bilateral chronic active pyelonephritis
- IV. Compensatory hypertrophy of left kidney (300 grams)
- V. Aortic atherosclerosis, minimal
- VI. Hemorrhagic cystitis
- VII. Exogenous obesity

JOURNAL OF MEDICINE. Today, Dr. Raszkowski and I will allow him a brief rest from this time-consuming but very important task. We want to use this forum to express our appreciation to our long time friend and mentor for all he has contributed to this medical community.

Our second purpose is to use the clinical information and autopsy findings of this case to explore the development of this complex area of medicine since the time Dr. Raszkowski and I first put together the fascinating autopsy data in 1970.

We did know about the Zollinger-Ellison syndrome and a complex disorder known as multiple endocrine adenomatosis in 1970. Since then we have learned how to routinely measure parathormone and gastrin, significantly improved routine serum calcium methodology and have changed our approach to gastric analysis quite dramatically. As usual the new tools have sometimes created new problems.

Serum gastrin levels became readily available to practicing physicians in our region in 1975. This hormone exists as a molecule consisting of 13, 17 or 35 polypeptide units, sometimes referred to as mini, little and big gastrin. The gastrin level when measured by RIA, consists of all three forms of gastrin. In the patient with a gastrinoma, bits and pieces of these molecules may also be recognized by the test system as gastrin contributing to the high assay level, but with no physiologic activity.

In the normal individual, gastrin secretion is stimulated by the flow of protein digestion products into the antrum of the stomach. Serum calcium also contributes to the quantity of gastrin release, but to a minimal degree. Gastrin is inhibited by a pH of less than 2.5, by secretin and through the neural cholinergic pathway. The net result of the interaction of

the stimuli and the inhibitors is to increase or to decrease hydrogen ion secretion by the parietal cells of the stomach. Other effects of gastrin include stimulation of growth of gastric mucosal cells and decrease in rate of gastric emptying. Gastrin does not act alone in stimulation of hydrogen ion secretion; histamine and acetylcholine contribute in a synergistic but less potent manner.

In the patient with a gastrinoma, gastrin produces significant hyperplasia of the gastric mucosa resulting in rugal hypertrophy, increased rate of gastric emptying, excessive acid production causing duodenal and jejunal ulcers plus lesser mucosal changes resulting in pain and inactivation of lipase and bile acids which may cause diarrhea. Concomitant hyperparathyroidism may greatly augment serum gastrin production in these patients.²

The gastric analysis is necessary to correctly interpret gastrin levels. We no longer speak of combined and free acid. The basal acid output/maximal acid output ratio of 0.6 or greater is less important now than what it was several years ago. The same is true for the 12 hour overnight aspiration of gastric contents. A basal acid output (BAO) of greater than 15 mEq/hr is characteristic of patients with gastrinoma. However, if reported series are combined, 32% of patients with gastrinoma have had a BAO of less than 15 mEq/hr. Only 10% of the duodenal ulcer patients, however, will have hydrogen ion secretion greater than 15 mEq/hr.

Interpretation of the gastric analysis becomes somewhat more difficult if the patient has had a previous acid reducing operation. The typical patient with gastrinoma will have a BAO of greater than 15 mEq/hr. Only 55% of patients meet this criterion.³

Our interpretation of gastrin, or perhaps a series of gastrin levels, since the value may fluctuate considerably in any given patient, is valid only after consideration of the gastric analysis data. The differential diagnosis for an elevated serum gastrin level with normal or decreased gastric acid output includes: pernicious anemia, chronic gastritis, gastric cancer, vagotomy, and for reasons I do not understand, pheochromocytoma.

In all of these conditions, however, the gastric analysis shows low levels of gastric acid and the high gastrin levels reflecting a physiologic increase in gastrin. When the gastric acid is significantly increased, the differential diagnosis has to include gastrinoma (Zollinger-Ellison syndrome), retained antrum syndrome, antral G cell hyperplasia, antral G cell hyperfunction. gastric outlet obstruction, massive small bowel resection and renal failure. It is necessary that each of these entities be taken into consideration. To differentiate them we can use the fasting serum gastrin, serum gastrin following a test

meal and serum gastrin level following an intravenous bolus of secretin. These studies should be ordered only after collection of the necessary clinical information. ^{8, 9} The test meal and secretin test must be carefully standardized and follow a strict protocol. They are expensive, and haphazard data collection could result in confusion or even be dangerously misleading.

Let us look at the most significant features of each of these disorders. ¹⁰

- 1. Gastrinoma (Zollinger-Ellison syndrome) The BAO will be greater than 15 mEq/hr in 70% of patients and all or nearly all will have increased fasting serum gastrin on one or more occasions with 50% of patients higher than 500 pg/dl. The serum gastrin increase is less than 50% over basal levels following a test meal. However, the secretin test will demonstrate a marked and paradoxical increase in gastrin levels. Benign or malignant neoplasms can be identified in about half of the patients.
- 2. Retained antrum syndrome The patient with this rare problem will have a history of Billroth II gastroenterostomy. Part of the antrum has been left attached to the excluded duodenal stump and can be demonstrated using the technetium pertechnetate scan. BAO may be greater than 15 mEq/hr and fasting gastrin levels are equivalent to those of the gastrinoma patient. The serum gastrin levels change little with the test meal and may decrease after injection of secretin.
- 3. Antral G cell hyperplasia The existence, incidence and clinical manifestations of this disorder are still subject to debate. The BAO is usually less than 15 mEq/hr. Fasting serum gastrin is mildly increased with greater than a 100% increase characteristic after the test meal and little or no change following injection of secretin.
- 4. Antral G cell hyperfunction This is frequently a familial disorder and may be associated with hyperpepsinogenemia I. BAO and fasting gastrin levels are generally not strikingly increased. The serum gastrin increase following the test meal is greater than 100% over fasting levels, but change minimally with the secretin test.
- Gastric outlet obstruction BAO and the fasting serum gastrin are mildly increased. Presumably this is secondary to decreased gastric emptying. Nasogastric suction will return fasting gastrin levels to normal.
- Massive small bowel resection The patient will have a history of surgical removal of a substantial portion of the small intestine. BAO and serum gastrin are mildly elevated and serum gastrin increases.

DR. RASZKOWSKI: The case for discussion to-

day is one that has held a fascination for me since I participated in the autopsy in February of 1970, as a part of the sophomore pathology course at this medical school. At the time of the final autopsy summary Dr. Ohrt noted that "with the autopsy findings the picture clears considerably," but this is often the case. This fact is pointed out in the recent article from Brigham and Women's Hospital in Boston where 10% of randomly selected autopsies from 1960, 1970, and 1980 revealed "a major diagnosis that, if known before death, might have led to a change in therapy and to prolonged survival." ¹¹

This case continued to hold my interest long after I completed the first two years of medical school in South Dakota and ultimately, I believe, was responsible for my career choice of gastroenterology. I believe it is of interest to look at what was known about selected aspects of this case in 1970 and to ask what has changed over the subsequent 13 years.

The association of peptic ulceration and endocrine tumors was first firmly established in 1955 by Robert M. Zollinger and Edwin H. Ellison. 12 These surgeons described two patients with the clinical entity, which now bears their names, consisting of marked gastric hypersecretion and hyperactivity, intractable peptic ulceration, and non-insulin producing (non-beta) islet cell tumors of the pancreas. Their report served to stimulate interest in the endocrine aspects of peptic ulceration 13 and while they believed the syndrome to be rare, in a little over ten years after their initial report more than 600 cases have been reported in the literature. 14

Gastric hypersecretion was noted to be the single most characteristic finding in patients with Zollinger-Ellison syndrome (ZES) and was present in 95% of the first 260 collected cases. Eighty-five percent of these patients had overnight (12 hour) gastric aspirations of more than 1000 ml and in 75% the free acid content of this overnight collection exceeded 100 mEq. The stomach was noted to be secreting hydrochloric acid at or near its maximum capacity, as four-fifths of these patients had little or no increase in HCl production following the administration of histamine.¹⁵

From 1955 to 1970 it was noted that the initial components of ZES needed to be expanded, as individual patients respond in different ways to excessive amounts of hydrochloric acid in the upper gastrointestinal tract. Although the presence of peptic ulceration in the second, third or fourth portion of the duodenum was suggestive of this syndrome, and ulceration in the proximal jejunum was felt to be almost pathognomonic for ZES, ¹⁶ approximately 60% of these ulcers were originally noted to occur in the duodenal bulb — prior to the patient's first ulcer surgery. ¹⁵ Thus, it became apparent that the most

usual initial presentation of this syndrome was that of a "routine" duodenal ulcer. Atypical ulceration was felt to be a secondary manifestation of surgical therapy and was present in approximately 75% of one collected series. ¹⁷ These ulcers might occur in any portion of the esophagus, stomach, duodenum, or upper jejunum, and multiple sites of peptic ulceration were noted in approximately 10% of these patients. ¹⁸

A watery diarrhea, so massive that it has been called "pancreatic cholera," occurred in approximately one third of all ZES patients and could precede or coexist with the peptic ulceration. The transit time from mouth to anus in these patients could be under one hour and the fluid and electrolyte loss so massive that patients could succumb to diarrhea and potassium loss before peptic ulceration was even noted. In other patients steatorrhea rather than diarrhea was noted as the primary manifestation of ZES. 20, 21

In the early 1960's it became apparent that the link between peptic ulceration and the non-beta islet cell tumors of the pancreas was the powerful gastric secretogogue, gastrin. This hormone was isolated from extracts of non-beta islet cell tumors from both primary and metastatic sites^{22, 23} and from the plasma and urine of patients with ZES.²⁴

In addition to an islet cell adenoma, which was originally described as an integral part of the syndrome, carcinoma and diffuse hyperplasia of the islet cells were subsequently noted to produce gastrin and thus produce ZES. These tumors could be either single or multi-focal and were not necessarily limited to the pancreas as such tumors were identified in the wall of the duodenum, the wall of the stomach, the hilus of the spleen, and in regional lymph nodes. ^{14, 25, 26}

In 1970 the preoperative diagnosis of ZES was dependent upon a high degree of suspicion as most patients were diagnosed only at the time of reoperation for recurrent ulceration following what was believed to be adequate surgical therapy for peptic ulcer disease. 16 Most patients were noted to be between 30 and 60 years of age and most had an initial presenting syndrome which was similar to that observed in benign peptic ulcer disease uncomplicated by pancreatic neoplasia. Epigastric pain was the most common complaint followed by nausea and vomiting, weight loss, diarrhea, and melena. The syndrome was characterized in the literature by its chronicity and in one series 80% of patients with ZES had been ill for one year while more than 25% had been ill for more than five years prior to diagnosis. 15, 17

The most consistent laboratory findings in ZES were those of an elevated basal acid secretion and a

lack of significant increase in acid secretion following histamine stimulation. However, gastrinomas may secrete only intermittently and a single gastric analysis was noted to often be deceptively normal.²⁷ It was noted in 1970 that in the near future the radioimmunoassay technique for serum gastrin would add much to the diagnostic armamentarium.¹⁴

Prior to the routine availability of serum gastrin assays, it is felt that the initial responsibility for the diagnosis of ZES was that of the radiologist. The roentgen findings were noted to reflect the altered physiology of the upper gastrointestinal tract rather than the anatomic presence of a pancreatic tumor. Marked enlargement of the mucosal folds of the stomach, duodenum and jejunum; megaduodenum; and increased intragastric fluid without obstruction coupled with typical or atypical peptic ulceration are highly suggestive of the ZES radiographically. ^{18, 28}

The proper surgical methods of treatment for both the pancreatic neoplasm and the peptic ulceration associated with ZES were widely discussed as there was no consistently effective medical treatment for this syndrome available until the mid-1970s.²⁹ The removal of the active endocrine tissue of the pancreas had been advocated but this was often impossible in small multiple tumors, hyperplasia, or malignant tumors with metastases. In addition the ulcer, rather than the neoplasm, was felt to be the most critical problem since these neoplasms are often slow growing. Therefore, it was generally agreed that the target organ, the stomach, should be removed in toto as the primary form of therapy. Retrospective studies indicated that total gastrectomy offered patients a decidedly longer survial. 16, 26, 30 It was also suggested during this era that a total gastrectomy had a beneficial effect upon the tumor and its metastases, as both appeared to regress following total gastrectomy. 31 In 1970 Zollinger reported a followup on one of his two initial patients. He stated that she:

"has had two children since a total gastrectomy, at five and seven years after operation. This woman had metastases to the regional lymph nodes but ten years after total gastrectomy I was unable to prove any evidence of metastasis at the time of cholecystectomy for gallstones. She is doing quite well fifteen years after surgery . . . this patient . . . has given us great courage to maintain our firm position favoring total gastrectomy." ³²

In addition to broadening the characteristics of ZES in the 1960s, it became apparent that this syndrome could be but a component of a familial disease transmitted by a dominant gene.³³ This syndrome was then called multiple endocrine adenomatosis

and by 1970 thirty family pedigrees had been reported in the literature.³⁴

Multiple endocrine adenomatosis was characterized as a familial disorder with the concomitant occurrence of multiple tumors or hyperplasia involving endocrine organs. The syndrome, as originally described by Wermer,³⁵ included: benign tumors of the anterior pituitary, adenomas or hyperplasia of the parathyroid glands, multiple benign or malignant islet cell tumors (either beta or non-beta), and peptic ulceration. To this, by 1970, had been added the less common components of: adenomatous goiter, multiple benign or malignant adrenal cortical tumors, pheochromocytoma, carcinoid tumor, and multiple lipomatosis. ¹⁴, ³⁴

It was recognized by 1970 that approximately 20% of patients with ZES had evidence of multiple endocrine adenomatosis. ¹⁵ The most frequent locations for endocrine involvement beyond the pancreas were the parathyroid glands and the anterior pituitary. ³⁶

Thus, by 1970 ZES was recognized as a potential component of a broader familial syndrome with the potential for multiple endocrine involvement. The ulcerogenic hormone in these patients, gastrin, had been identified and there was a wide ranging discussion in the literature about the ulcerogenic potential of increased levels of other hormones from endocrine glands which might be involved. The potential familial pattern in ZES was stressed and both immediate and long term screening of family members was advocated. ³⁶, ³⁷

Although the clinical picture in ZES remains unchanged, in the ensuing 13 years from 1970 and 1983, many things have changed which might have altered the outcome in this case. Before turning to some of these, it is important to mention that the terminology has changed in one area. With the study of additional kindreds, multiple endocrine adenomatosis had become multiple endocrine neoplasia and has been subdivided into Type I, Type IIA, and Type IIB (III). The components of these syndromes are listed in Table II.

Major changes have occurred both diagnostically and therapeutically which radically alter the approach to a patient with ZES today. Diagnostically, the 12-panel has become a routine part of the screening laboratory evaluation and parathormone and especially serum gastrin determinations have moved from research tools to readily available assays. Endoscopy now allow visualization of all portions of the bowel except the small intestine and new imaging techniques such as ultrasound and computer tomography have greatly added to the ability to diagnose intraabdominal disease.

The treatment of peptic ulcers in general, and the

TABLE II

Components of Multiple Endocrine Neoplasia Syndromes

Type I

Hyperparathyroidism
Pancreatic tumors
Zollinger-Ellison syndrome
Insulinoma
Glucagonoma
WDHA syndrome*
Pituitary tumors
Nonfunctioning
Acromegaly
Cushing's syndrome
Prolactinoma
Lipomas
Adrenal nodules
Thyroid nodules

Type II Medullary thyroid carcinoma Pheochromocytoma Hyperparathyroidism

Bronchial carcinoid tumors

TYPE IIB (TYPE III) Medullary thyroid carcinoma Pheochromocytoma Multiple mucosal neuromas Marfanoid habitus

* WDHA — (Watery diarrhea hypokalemia achlorhydria)

treatment of ZES in particular have changed remarkably in the last 13 years. Not until 1977 were antacids proven to be effective therapy for duodenal ulcer disease and this was in the face of several previous studies showing no statistical benefits in duodenal ulcer therapy with antacids over placebo. ³⁸

It was not until the early 1970s that the first selective histamine₂ (H₂) blocking agent was reported and in 1977 cimetidine (Tagamet) was introduced. Cimetidine offered an alternative pharmacologic approach for duodenal ulcer patients and revolutionized the therapy for ZES. Many patients can be managed medically with cimetidine (often at dosages significantly higher than for duodenal ulcer disease) and anticholinergics rather than by total gastrectomy. More recently, a second generation H₂ blocker, ranitidine (Zantac) has been shown to be an effective pharmacologic therapy for patients with ZES.¹⁰

Some patients with ZES may still require surgery. Highly selective vagotomy may make medical management with an H₂ blocking agent more effective³⁹ and a total gastrectomy may be required in patients who do not conscientiously adhere to medical therapy.⁴⁰ However, peptic ulceration can be con-

* General Surgeon, Sioux Valley Hospital and faculty, School of Mcdicine, University of South Dakota, Sioux Falls, SD.

trolled in most patients by an H₂ blocker alone or in combination with an anticholinergic agent. Like total gastrectomy, this therapy has no effect on tumor growth and the beneficial effects are entirely due to the control of gastric secretion.

Today, the diagnosis of ZES can usually be eliminated prior to surgery for peptic ulcer disease by the use of fasting serum gastrin determinations. This syndrome should no longer be a disease which is only suspected subsequent to a stormy postoperative course for peptic ulcer disease and the use of H₂ blocker therapy has revolutionized the management of these patients.

As I reviewed the diagnostic and therapeutic changes which have occured in the past thirteen years, I believe that the outcome in this case may have been very different if the patient was hospitalized in 1983 rather than in 1970. Even the final pathologic diagnosis may have been different. The diagnosis of multiple endocrine neoplasia-Type I requires a positive family history⁴¹ which was lacking in this case. Also, parathyroid hyperplasia has recently been suggested to be secondary to ZES or its metabolic consequences rather than a separate and distinct component of multiple endocrine neoplasia — Type I.⁴¹

One thing which has not changed over the years is the commitment of Dr. John Barlow to quality in education and in health care. It is an honor for me to participate in this clinical pathological conference as we express to you, John, our appreciation for the years you have given to us as our teacher, our colleague, and our friend.

DR. OHRT: I would like to call on Dr. Arneson for comments as he operated on this patient in 1970. It has to be realized that with our modern techniques this disease is easier to diagnose than it was back in 1970.

DR. WALLACE A. ARNESON*: First, I would like to say that I always try to forget the bad results, as all good surgeons do. In that way, I can say unflinchingly that, to the best of my knowledge, I have never lost a case. As Dr. Kendall Burns and I operated on this case and both of us have had some training under Dr. Zollinger, he was not too pleased when we told him about how we had missed the diagnosis of the only syndrome named after him. DR. AUSTIN L. VICKERY†: I am very impressed with the thoroughness of the discussion. I would like to point out that most of the cases of ZES are due to malignant gastrin producing tumors of the pancreas whereas other well-known syndromes such as insulin secreting pancreatic tumors, are usually caused by benign adenomas.

DR. GILES TOLL‡: It was a pleasure to hear this

[†] Professor of Pathology, Harvard Medical School; Pathologist, Massachusetts General Hospital, Boston, Massachusetts.

[‡] Pathologist, Presbyterian St. Luke's Hospital, Denver, Colorado.

articulate and scholarly discussion. We have been honored to have a number of graduates from your medical school training as residents in Denver. Their quality of training and conduct has been exemplary, but I would like to clarify one point. It was mentioned once to me that Dr. Barlow's nickname of 'The Bear' was given to him by the University of South Dakota students. Dr. Wegner and I think it was given to him during our residency together, but I assure you it was well in place before he came to South Dakota.

DR. KARL H. WEGNER*: I would like to call on Dr. Robert VanDemark, Jr., M.D., since his father could not be here to speak in behalf of the SOUTH DAKOTA JOURNAL OF MEDICINE.

DR. ROBERT VANDEMARK, JR.†: My father regrets not being able to be here because he was out of town. I think it is a tribute to Dr. Barlow for all he has done for the SOUTH DAKOTA JOURNAL OF MEDICINE. Looking around the room and seeing all your former students and colleagues, I can only second what has been said.

DR. WEGNER: I would like now to reintroduce Dr. Vickery who will make some comments.



JOHN F. BARLOW, M.D.
APPLICATION FOR APPOINTMENT
AS RESIDENT, MASSACHUSETTS
GENERAL HOSPITAL, JULY 1959

Figure 1

† Orthopedic Surgeon, Faculty, School of Medicine, University of South Dakota, Sioux Falls, SD.

DR. VICKERY: Although this occasion is a tribute to John Barlow's stewardship of 17 years of CPC's, I believe this is just one facet that derives from his thirst for knowledge and enthusiasm for medicine. His mentors and colleagues have always appreciated his drive and numerous accomplishments.

I did some investigation of our files and obtained the following data. Here is a photograph on his application in July 1959. (Fig. 1). Despite the shy and retiring personality and fragile physique, he was appointed as third assistant resident. Here is the letter of confirmation (Fig. 2). This letter also documents the fact that, in spite of widespread misconception, the administration at Harvard Medical School believes that appointment to their staff should not be tarnished by any substantial financial rewards.

It is the custom to give the residents every opportunity to get right to work and I looked up his first autopsy. These are the final anatomic diagnoses. (Fig. 3).

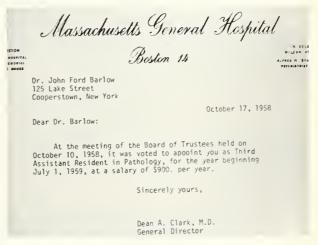


Figure 2

Necropsy No. 22,077 July 3, 1959 at 5 p.m.	WM: 852206 Age 57	
21½ hours post mortem	Prosecutor: J. F	

ANATOMIC DIAGNOSES

Chronic pyelonephritis, bilateal, severe with (uremia).	19-100.0 (551)
Esophageal stricture, lower third, benigh.	6374
Malnutrition, severe, with emaciation.	014-711
Operation: Paraesophageal haitus herniorrh	ару
and biopsy of esophagus, 8 months.	350-4963
Pulmonary atelectasis, lower lobes, bilateral	,
due to aspiration.	362-yx4
Bronchopneumonia, focal bilateral, acute.	361-190
Osteomalacia.	200-9.9
Osteitis fibrosa cystica of bone, minimal	200-773
Parathyroid hyperplasia, secondary mild.	830-7x6
Fatty vacualization, moderate, liver	680-701

Figure 3

^{*} Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, former Dean, School of Medicine, University of South Dakota, Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

You will note this was a lady with uremia from chronic pyelonephritis. Secondary parathyroid hyperplasia was found indicating that parathyroid dissection has been performed. At this early stage John's characteristic thoroughness was already in evidence.

I also asked several other members of his peer group if they could remember any stories. The most common one was that he was the only person at Dartmouth who got caught breaking into the library to study. One other was that his wife, Anne, finally got him to take a vacation and he promptly packed a microscope and some slides to take to Cape Cod with them. One former colleague asked "Tell me, how is his handwriting? Does he still use a pen like it is a fencepost?"

I have a telegram to you, John, from one of your old mentors, Dr. Robert E. Scully who succeeded Dr. Castleman as the editor of the CPC's in the New England Journal of Medicine.

"John Barlow, M.D., Member No. 3

Congratulations and condolences for starting your eighteenth year of CPC's in the SOUTH DAKOTA JOURNAL OF MEDICINE. Only those of us who have similarly suffered can appreciate what you have accomplished. By the way, have you got that case ready for the Journal tomorrow?

> Bob Scully Secretary of International Society of CPC Editors Unincorporated''

John, I would now like to present to you a bound volume of the CPC's for the last ten years.

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Minnesota Medical Association Resource Group on **Rheumatic Diseases**

RHEUMATOLOGY SEMINAR V March 6-March 13, 1984

LOCATION: Paradise Grant Hotel, Nassau, BAHAMAS

DATES: Departure from Twin Cities Airport on Tues-

day, March 6

Return to Twin Cities on Tuesday, March 13 Educational Program — March 7-11

FEE: \$285 (educational program)

Approximately: \$1378 per physician/\$324 per accompanying spouse or child (includes round-trip flight, ground transportation and accommodations for seven nights)

AUDIENCE: Primary care physicians and physicians who are involved in the care of arthritic patients.

FACULTY: From the University of Minnesota and the

Mayo Clinic

CONTENT: Common rheumatologic problems, diagno-

sis, treatment and the course of the disease

HOURS: 20 hours, Category I/Prescribed

CONTACT: Department of CME and Meeting Services,

Minnesota Medical Association, Suite 400, 2221 University Avenue SE, Minneapolis,

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S Auxiliary News

At the request of Dr. Joe Hamm it was my privilege to be a guest at the SDSMA Executive Commission meeting in August. Auxiliary plans for 1983-84 were briefly outlined.

Mary Barrett has been appointed AMA-ERF chairman to replace Dorothy Hamm who is recuperating from heart surgery. AMA-ERF began accepting donations to the new Medical Student Assistance Fund in January, 1983.

At the national convention in June, South Dakota's District 1 received the North Central Regional AMA-ERF award for largest contribution per capita for the second consecutive year.

Mary Cosand, legislation chairman, has been appointed an ex-officio member of the medical association's Commission on Legislation and Governmental Relations for 1983-84. Mary is in the process of making arrangements for the annual "Legislative Day in Pierre." This will be held in conjunction with the winter SoDaPAC meeting and the third auxiliary board meeting. All South Dakota physicians and spouses are reminded to invite their legislators to an informal evening of cocktails and dinner.

Top priority among auxiliaries and medical associations is the recruiting and retaining of members. Mollie O'Krafka has stimulating plans for South Dakota district chairmen to implement.

Once again we are focusing on the "Sponsor a Spouse" program and encouraging medical student and resident physician spouses to join our ranks.

Karen Pekas, health projects chairman, is continuing with the national Shape Up for Life campaign. Over the last five years we've been encouraging people to take notice of proper diet, exercise, stress management, and substance abuse.

At the request of the AMA, the promotion of awareness and prevention of child abuse will be one of the focuses of the AMA and South Dakota auxiliaries during 1983-84. As a result of this request, Charles Pelton, M.D., was asked to address the auxiliary Summer Conference on the subject of Child Abuse and Neglect.

Other conference speakers on August 11 were Betty Szewczyk, North Central Regional Vice-President; Ila Lushbough, National AMA-ERF Committee; Guy Edwards, A. G. Edwards & Sons, Inc.; and Marilyn Gelhaus, Insurance Counselor.



Dr. Hamm and each Medical Association district secretary will receive copies of the Auxiliary Newsletter this year from Jacquelyn Gunnarson, newsletter editor.

Thanks to the Association for support of the auxiliary, our Newsletter, and for this journal space enabling us to contact state physicians.

Auxiliary members are available to provide service to the SDSMA by coordinating and promoting your programs, priorities and goals.

When necessary, we are also available to provide manpower to respond to legislative issues affecting health care and medical practice.

Marie Howland

Marie Hovland, President South Dakota State Medical Auxiliary

S Department of Health

FETAL ALCOHOL SYNDROME — INTRAUTERINE CHILD ABUSE

Fetal alcohol syndrome is believed to be the second most frequent birth defect in the United States and the number one cause of mental retardation. Fetal alcohol syndrome is zero percent curable, expensive to care for, but 100 percent preventable.

The angel admonished Samson's mother, Judges 13:7, "behold thou shalt conceive and bear a son and now drink no wine or strong drink." In ancient Carthage newly wedded couples were forbidden to drink alcoholic beverages. During the "gin epidemic' in England in the early 18th Century, James Sedgewick noted, "half the train of chronical diseases will be brought on infants by the debauchory of the mother." In 1834 a report in Britian described "a starved, shriveled and imperfect look" of infants born to alcoholic mothers. In the first half of the 20th Century conflicting reports suggested there was an association between maternal drinking and fetal anomalies and others documented these findings as "superstitions." Surprisingly it has been only in the last ten years that general attention and concern has again been focused on the fetal alcohol syndrome.

Alcoholism is without clean cut diagnostic criteria; however, fetal alcohol syndrome has a clearly defined set of symptoms and signs. Central nervous system dysfunction, growth deficiencies, facial abnormalities and other variable malformations make up the syndrome of Fetal Alcohol Syndrome (FAS). Central nervous system effects are microcephaly, mental retardation and fine motor dysfunction. There is prenatal growth deficiency with birth length and birth weight well below normal. Growth deficiency continues throughout the postnatal period and there is no catch-up growth. Facial abnormalities include short palpebral fissures, microphthalmia and increased epicanthal folds (mongol slant). There is under development of the maxillae with resultant broad nasal bridge, flattened contour of the midface and protruding chin. Cleft lip and palate and low set ears are of common occurrence. Other frequently associated malformations are cardiac septal defects, hirsutism, hemangiomas, poor muscle tone, genital defects and a multiplicity of limb and skeletal abnormalities. There may also be a less pronounced affect of alcohol on the fetus resulting in fetal alcohol effect (FAE). This may be manifest by borderline changes in facial characteristics and growth delay. Central nervous system changes may be reflected in hyperactivity.

The exact mechanism of cause and effect is not yet known. It is uncertain whether the damage is a direct toxic affect of alcohol or of the metabolites of alcohol. The defects and growth deficiency may be the result of a reduced number of fetal cells. The effect upon the fetus may be dose related. A high blood alcohol level during a critical time in fetal development may be as devastating as high alcohol intake throughout pregnancy. This presents the frightening prospect that the damage may take place when the woman does not know that she is pregnant. Fetal cells and tissues may be unable to tolerate a toxic chemical environment, when in a certain stage of development, for even a few hours. Results of some animal studies indicate that paternal alcohol consumption prior to conception may have an adverse effect upon the developing fetus. At present these data are not conclusive.

Recent studies suggest that the incidence of fetal alcohol syndrome (FAS) may be between one and two per thousand live births. There may be a fetal alcohol effect (FAE), or partial expression of the syndrome, in as high as three to five per thousand live births. There were 12,839 live births in South Dakota in 1982. This means that there is the possibility that there were 12 to 24 infants born with fetal alcohol syndrome and that 36 to 60 infants will show fetal alcohol defect.

The Department of Health, in 1983, added the item, fetal alcohol syndrome, to the certificate of live birth. This was added in order to increase awareness of the syndrome and as an attempt to encourage reporting so more valid statistics may be obtained.

Using figures provided by the Office of Developmental Disabilities, yearly cost estimates for care for each case of fetal alcohol syndrome could range from \$10,000 to \$30,000. This would mean that with present incidence estimates the yearly cost for new cases of FAS could range from \$120,000 or \$360,000 to \$240,000 or \$720,000. This does not include the cost of additional medical and educational expense of the 36 to 60 infants born with fetal alcohol effect. All of these costs multiplied by a 60 year life expectancy run into astronomical sums.

The burden of the control of this avoidable, irreversible and devastating syndrome is in prevention. The key to this control, lies not only with the mother, but with the family and the community in understanding the risk of alcohol for the newborn child. The physician has a responsibility in determining risk factors, providing information and counseling, both before and during pregnancy, and in recognizing the infant with fetal alcohol syndrome.

Willis F. Stanage, M.D.
John B. Gregg, M.D.
Lawrence J. Massa
Office of Medical Services
South Dakota Department of Health

S Future Meetings

November

- The John I. Coe Pathology Symposium: Computerization in Anatomic Pathology and New Horizons in Immunodiagnostic Techniques, Pillsburgy Aud., Hennepin Cty. Med. Ctr., Minneapolis, MN, Nov. 3. Fee: \$100. 6 hrs. Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Current Concepts in Head and Neck Pathology E.T. Bell Fall Pathology Symposium, Hennepin Cty. Med. Ctr., Minneapolis, MN, Nov. 4. Fee: \$120. 6 hrs. Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Implications of DRG Reimbursement for Hospital-Physician Relations, Sheraton Inn, Aberdeen, SD, Nov. 9. Contact: Kathy Graham. Phone: 229-4040.
- Implications of DRG Reimbursement for Hospital-Physician Relations, Holiday Inn, Mitchell, SD, Nov. 10. Contact: Sue Wermers. Phone: 996-6501.
- Clinical Strategies in Primary Care Medicine, St. Paul-Ramsey Med. Ctr., St. Paul, MN, Nov. 10-12. 18 hrs. Category I credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- American Association for Cancer Education: Education in Preventive Oncology 17th Annual Meeting, U. of Minn., Minneapolis, MN, Nov. 17-18. Fee: \$120. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.
- Family Violence in the Deaf Community, St. Paul-Ramsey Med. Ctr., St. Paul, MN, Nov. 30-Dec. 1. 12 hrs. Category I credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

December

- Frontiers in Medicine, St. Joseph's Hosp., St. Paul, MN, Dec. 3. 7 Hrs. Category I credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, Dec. 7. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Coronary Heart Disease: A Comprehensive Review of Principles and Practice, St. Paul-Ramsey Med. Ctr., St. Paul, MN, Dec. 7-10. 18-62 hrs. Category 1 credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Pediatric Advanced Life Support, Des Moines, IA, Dec. 9-10. AMA Category 1 credits. Contact: Richard M. Caplan,

- M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- American Cancer Society National Conference on Advances in Cancer Therapy, New York, NY, Dec. 8-10. 16.5 hrs. AMA Category I & Am. Osteo. Assoc. credits and 15 hrs. AAFP credits. Contact: Nicholas G. Bottiglieri, M.D., Advances in Cancer Therapy Conf., Am. Cancer Soc., 777 3rd Ave., New York, NY 10017.
- Ear, Nose and Throat Diseases in Children: A 1983 Update, The Breakers, Palm Beach, FL, Dec. 10-14. Fee: \$250. 17 hrs. Category I credits. Contact: Dept. of Otolaryngology, Children's Hosp. of Pitts., 125 DeSoto St., Pittsburgh, PA 15213. Phone: (412) 647-5465.

January

- Sports Medicine Symposium, U. of Iowa, Iowa City, IA, Jan. 5-7. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- 2nd Biannual Gastrointestinal & Hepatic Diseases Conference, Mauna Kea Beach Hotel, Hawaii, Jan. 16-20. 20 hrs. Category I credits. Contact: Yvonne Brewer, M.P.H., Ed. Dir., Honolulu Med. Group Res. & Ed. Found., 550 S. Beretania St., Honolulu, HI 96813. Phone: 808-537-2211, ext. 751.
- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, Jan. 19. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- 8th Annual Hawaii Echocardiography Conference, Kahala Hilton Hotel, Honolulu, Jan. 23-27. 20 hrs. Category I credits. Contact: Yvonne Brewer, M.P.H., Ed. Dir., Honolulu Med. Group Res. & Ed. Found., 550 S. Beretania St., Honolulu, HI 96813. Phone: 808-537-2211, ext. 751.
- Third Annual Winter Congress in Medical Diagnostic Imaging, Chamonix, France, Jan. 26-Feb. 4. Fee: \$360. 26 hrs. Category I credits. Contact: Secretary, Winter Congress, West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (213) 340-0580, ext. 280.

February

Winter Seminar in Medical Diagnostic Imaging, London, England, Feb. 4-8. Fee: \$275. 15 hrs. Category I credits. Contact: Secretary, Winter Congress, West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (213) 340-0580, ext. 280.

March

St. Moritz 1984: Advances in Diagnostic Imaging, Palace Hotel, St. Moritz, Switzerland, Mar. 24-Apr. 1. 20 hrs. Category I credits. Contact: Educational Symposia, P. O. Box 17241, Tampa, FL 33682. Phone: (813) 971-6000, ext. 1112.

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Medicine

Clinicopathological Conference Fifty-Six Year Old Caucasian Female With Infection of Knee and Acute Renal Failure

Pemphigus: A Review of Current Concepts

Table of Cantents: page 3

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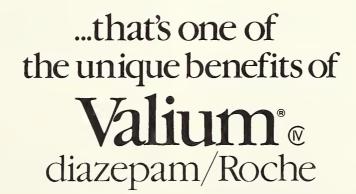
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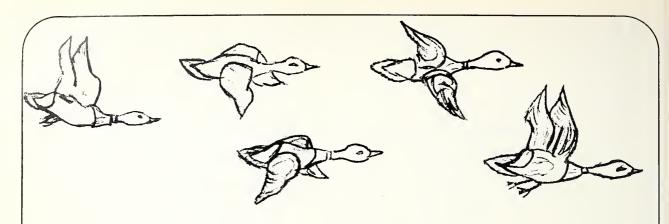
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We Extend Best
Wishes For A
Happy Thanksgiving



S Clinicopathologic Conference

Fifty-Six Year Old Caucasian Female With Infection of Knee and Acute Renal Failure

Robert C. Ulrich, M.D.* Richard A. Jaqua, M.D.† Discussers

John F. Barlow, M.D.‡ Editor

Case #940 712

This 56 year old Caucasian female entered Sioux Valley Hospital because of pain in the right knee.

Approximately eight months prior to admission, the patient was admitted for the first of two surgical procedures to correct degenerative joint disease of the knees. The left knee

was operated on first.

Approximately six months later she returned to the hospital and surgery was performed on the second knee. Both procedures were proximal tibial osteotomies. The first surgical procedure went without difficulty and healed well with marked improvement of the patient's problems. The patient had had a previous diagnosis of hypothyroidism for which she had intermittently taken thyroid hormone. She had a history of mild hypertension and had been placed on methylclothiazide. Blood pressure was approximately 154 systolic and 58 diastolic. She was restarted on thyroid medication when she was found to have ceased taking thyroid hormone at home.

Approximately two weeks following the second surgical procedure, the patient returned with severe pain in the right knee radiating down the leg into the foot. A large quantity of staphylococcus aureus coagulase positive, susceptible to most antibiotics but resistant to ampicillin and penicillin, was cultured from the proximal tibial osteotomy wound. The patient was placed on intravenous cephapirin. She responded well to therapy and was discharged on oral cephradine 200 mg orally four times each day.

The patient returned two weeks later because of recurrent pain and swelling of the knee and with increasing drainage.

She was seen in the emergency room, re-evaluated, and admitted. Staphylococcus was again cultured from the wound. She maintained that she had been taking her antibiotics as directed. She was admitted to the hospital and begun on intravenous cephapirin, one gram every 6 hours. She initially had an excellent response to that drug with decreased pain, swelling, and drainage. She was begun on sulfamethoxazole as well for urinary tract symptoms and hematuria. The thiazide diuretic and thyroid medication were restarted at this time by the attending physicians when it was discovered she had stopped taking these medications at home.

Approximately four to five days after admission, the patient developed significant pruritus. In addition, she developed a maculopapular rash on the back. The cephalosporin was stopped at that time and the patient was switched to a semisynthetic penicillin. The pruritus receded but the rash continued. Approximately two to three days later, it was noted that she had some mild azotemia. The semisynthetic penicillin was stopped and she was switched first to erythromycin and then to trimethoprim sulfamethoxazole. She continued to develop azotemia over the next several days.

PHYSICAL EXAMINATION: Temperature 37°C, pulse 84/min and regular, respirations 20/min and regular, blood pressure 178 systolic and 76 diastolic. There was a maculopapular rash noted over the arms, legs and torso. There were no pustular or bullous lesions noted. There was some mild periorbital edema. There was a perforated right tympanic membrane. There was a soft, apical, blowing systolic grade II/VI cardiac murmur. There was no obvious cardiomegaly. The heart had a regular rate and rhythm without gallops or rubs. The chest was relatively clear to auscultation and percussion. The abdomen was obese. There were no palpable organs, masses, spasm or tenderness. Pelvic and neurologic examinations were negative.

LABORATORY DATA: Urinalysis — yellow and hazy, pH 5.0, specific gravity 1.014, 3 + protein, negative for glucose, ketone bodies and bile, large amount of hemoglobin; sediment 20-25 wbc/hpf, 50-60 rbc/hpf. Initial urine culture showed no growth at 48 hours but one to two weeks later

^{*} Resident in Family and Community Medicine, Sioux Falls, SD. Affiliated with USD School of Medicine.

[†] Professor and Chairman, Dept. of Laboratory Medicine, USD School of Medicine; Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.

[‡] Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD

grew enterobacter cloacae and staphylococcus coagulase negative. Urinalysis over the hospital course continued to show 3-4+ proteinuria, marked microscopic pyuria and hematuria with white cell casts, coarsely granular casts and hvalin casts. No red blood cell casts were noted. Hemoglobin 10.5 gm/dl, hematocrit 34 vol/dl with normal red cell indices, total leukocyte count 9,900/mm³, (9.9 x 10⁹/L), with a differential count of 78% segmented neutrophils, 3% neutrophilic bands, 1% basophils, 3% eosinophils, 11% lymphocytes and 4% monocytes; reticulocyte count 1.1%, erythrocyte sedimentation rate 138 mm/hour. The hemoglobin dropped to a nadir of 8.3 gm/dl, but was restored to 11.5 gm/dl by transfusion. There was mild to moderate transient eosinophilia up to 7%. The erythrocyte sedimentation rate gradually dropped to normal by discharge. Three blood cultures were negative. Initial wound cultures of the leg were negative, but another grew staphylococcus coagulase negative. Admission blood urea nitrogen and creatinine were normal. These increased to 75 mg/dl and 8.0 mg/dl. Creatinine clearance was 14 cc/min (normal 105-150 cc/min). Twenty-four hour urine protein was 1.5 gm/24 hours; serum sodium 133 mEq/L, potassium 5.8 mEq/L, chloride 105 mEq/L, CO₂ content 20 mm/L. Arterial blood gases — pH 7.4, PCO₂ 34 torr, CO₂ content 22 mm/L, PO₂ 60 torr, O₂ saturation 89%, total protein 6.8 gm/dl with 0.4 gm/dl alpha-1 globulin, 0.8 gm/dl alpha-2 globulin, 1.0 gm/dl beta globulin, 2.0 gm/dl gamma globulin. The gamma globulin was slightly elevated with a broad or polyclonal pattern. Prothrombin time and partial thromboplastin time were within normal limits. Initial T₄ (thyroxine) was 1ug/dl; T₃ (triiodothyronine) 24 ug/dl and thyrotrophin level (TSH) over 100 IU/L. The patient had not been taking the thyroid hormone and these values returned to normal with thyroid hormone therapy. A fluorescent anti-nuclear antibody test (FANA) was negative. C₃ was 49 ug/dl (normal 56-120 ug/dl) and C₄ 45 ug/dl (normal 20-50 ug/dl). The alkaline phosphatase was 185 IU/L (normal 0-115 IU/L). This remained mildly elevated on repeat examination. Inorganic phosphorus was 5.1 mg/dl (normal 2.1-4.5 mg/dl), calcium 7.2 mg/dl (normal 8.4-10.7 mg/dl). The lactic dehydrogenase (LDH) was mildly elevated on one occasion. Aspartate aminotransferase (SGOT, AST), total bilirubin and glucose were within normal limits. Uric acid was 11.1 mg/dl on admission, but dropped to 5.7 mg/dl by discharge. Antistreptolysin 0, antidesoxyribonuclease B and antihyaluronidase titers were within normal limits. X-ray of the right knee including tomogram showed radiolucencies consistent with infection and soft tissue swelling about the osteotomy site. A bone scan showed increased uptake in this region. Ultrasound and computer tomogram studies showed the kidneys were normal in size and position.

The patient failed to respond to conservative therapy. An open renal biopsy was performed.

DR. ULRICH: There are multiple possibilities for the etiology of this woman's precipitous renal failure. However, the events surrounding the acute episode are very suggestive of an acute allergic interstitial nephritis. This is an immunologic phenomenon thought to be a delayed hypersensitivity reaction. Common medications implicated include: penicillin and its derivatives such as penicillin G or semisynthetic penicillins such as methicillin, nafcillin and finally thiazide diuretics. Previous exposure to these medications usually without an adverse reaction is commonly seen. There is usually a latent period before the onset of symptoms of 5 days to 5 weeks. The entity does not appear to be related to the dose of medication. The disease is reversible when the drug is withdrawn. However, supportive care such as

hemodialysis may be required in as many as 35% of the cases. A short course of steroid therapy to hasten recovery of renal function may also be necessary. Interstitial nephritis often presents with fever, rash and arthralgias. Abnormal urinary findings consist of mild to moderate proteinuria (80% of cases), pyuria (80% of cases), eosinophiluria (80% of cases) and microscopic hematuria (90% of cases). Occasionally gross hematuria may occur (30% of cases). The peripheral blood smear reveals a transient eosinophila in 60-80% of the cases. This is a very helpful positive finding provided no other causes for eosinophilia are found. The serum IgE may also be elevated in 50% of biopsy proven cases of drug induced interstitial nephritis.

This woman developed a maculopapular rash and arthralgias 4 to 5 days after an intravenous cephalosporin. She did have a history of previous penicillin allergy and usage of cephalosporin previously. She also had taken a thiazide diuretic which may also cause interstitial nephritis. In addition, the patient developed acute onset of renal failure with hematuria and pyuria, and demonstrated transient eosinophilia. Although a drug etiology is certainly likely, we cannot rule out immune complex disease secondary to the bone infection or some other etiology. Acute renal failure can be caused by hypotension or sepsis with renal ischemia. However, the history does not suggest this. Toxic nephropathy is also possible, but there is often a very rapid recovery following the removal of the offending agent.

With the history of degenerative joint disease, one can suspect that she has taken a significant quantity of analgesics over the years. As a matter of fact, talking to the clinician who attended this case, there was such a history. Recent literature suggests that 20% of cases of interstitial nephritis are related to analgesic drug abuse. The drugs most frequently indicated include phenacetin of which the active metabolite is acetamenophen, aspirin and many of the newer antiinflammatory agents. It has been suggested that the combination analgesic medications have a synergistic effect. In analgesic nephropathy, the striking histologic finding is renal papillary necrosis in addition to interstitial nephritis. However, there is no evidence of papillary necrosis in this case. Prior to the patient's acute episode of renal failure, she did have a six month history of asymptomatic pyuria and hyperuricemia which would support analgesic intake. However, the blood urea nitrogen and creatinine were normal although these parameters might not become significantly elevated until a considerable portion of renal function was lost.

It may be that she had some underlying interstitial abnormalities which predisposed to her acute episode. Chronic interstitial nephritis can be seen in nephrosclerosis secondary to hypertension or secondary to uric acid sludging and intrarenal tubular obstruction, renal obstruction secondary to stone or even renal tuberculosis.

In spite of the above I will stay with my original diagnosis of drug induced allergic interstitial nephritis.

Dr. Ulrich's Diagnosis Interstitial Nephritis Secondary to Drug Therapy, Probably the Cephalosporin

DR. JAQUA: Light microscopy showed renal cortex with generalized patchy tubular interstitial disease. This was characterized by an infiltrate of lymphocytes, histiocytes and plasma cells with active invasion and destruction of renal tubules (Fig. 1). There were a few eosinophils and neutrophils present in the infiltrates but these were not plentiful. PAS (periodic acid Schiff) and methenamine silver stains confirmed the tubular destruction and the Masson trichrome stain showed minimal associated fibrosis suggesting an acute process.

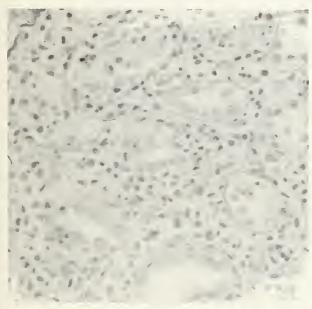


Figure 1
Extensive mononuclear interstitial infiltrate with invasion of tubule walls (mid portion of picture) H & E 100X

The majority of the glomeruli were uninvolved. However, a small fraction (in the range of 5%) showed varying degrees of parietal epithelial proliferation. These small crescents seemed to be associated with areas of significant interstitial inflammation. A rare glomerulus showed a very small segmental area of involvement characterized by capillary collapse and increase in mesangial matrix.

This pathology puts us in a diagnostic category of the lymphohistoplasmocytic type of interstitial nephritis. One can further subclassify this diagnostic category by immunofluorescence findings. It has been found that a number of the interstitial nephritides have a basis in humoral immunity and may be associated with linear or granular deposition of immunoglobulin and complement components along tubular basement membranes. The immunofluorescent studies on this biopsy were negative and this was confirmed by electron microscopic studies which showed no evidence of dense deposits.

The lymphohistoplasmocytic type of interstitial nephritis with negative immunofluorescence is not at all specific, but has been described most frequently associated with microbial infection, vaccination, drugs, glomerulonephritis, vasculitis, transplant rejection, etc. The clinical picture in this case would clearly implicate a drug reaction, antibiotics being the most likely. Table I gives a list of the antibiotics and other compounds implicated in the cause of acute tubular interstitial disease.

As you can see, the patient we are discussing today received several of the implicated antibiotics in the appropriate time interval. Most of the antibiotic agents implicated are thought to involve immune mechanisms. Although we could not demonstrate any humoral mechanisms there are other studies available such as lymphocyte transformation studies, etc., which will demonstrate the cellular immune nature of the process and implicate a specific antibiotic on some occasions.

TABLE I

Some Causes Associated with Acute Tubulointerstitial Nephritis

- I. Acute bacterial pyelonephritis
- II. Drug
 - a. Phenacetin (analgesic nephritis) often associated with papillary necrosis
 - b. Acute drug induced hypersensitivity reaction Methicillin

Oxacillin

Ampicillin

Carbenicillin

Rifampin

Cephalothin Cephalexin

Phenindione

Thiazides

Allopurinol

Furosemide

Phenytoin

Fenoprofen

III. Nonspecific acute (associated with infection)

Streptococcal disease

Diphtheria

Toxoplasmosis

Brucellosis

Staphylococcal disease

Viral disease

Legionaires disease

IV. Idiopathic

Note: Other drugs including various penicillins and cephalosporin antibiotics continue to be reported

FINAL ANATOMIC DIAGNOSIS INTERSTITIAL NEPHRITIS, PROBABLY DRUG INDUCED

DR. BARLOW: I had this case presented because drug induced interstitial nephritis is a common cause of acute renal failure and must be recognized quickly or irreversible renal damage will occur if the drug is continued.

DR. BRUCE VOGT: * Do you feel this patient had chronic interstitial nephritis with superimposed acute disease?

DR. ULRICH: I feel this was mainly an acute process at least clinically. There may well have been a chronic component.

DR. JAQUA: The histopathology also suggested an acute process.

DR. VOGT: As the pathology has nicely illustrated mainly chronic inflammatory cells, why do these patients have pyuria?

DR. JAQUA: There is pyuria in over 80% of patients in the acute interstitial nephritis related to drugs despite the interstitial infiltrate of lymphocytes and plasma cells of the delayed immune response which is often present. There are a number of conditions which give "sterile pyuria"; most prominent of which are viral, chlamydial infection and tuberculosis. The presence of pyuria itself does not point to a specific etiology but if a significant percentage of the leukocytes and eosinophils are present, this is helpful in implicating an allergic reaction.

DR. PLUMMER:† Could this patient have had a needle biopsy of the kidney as a diagnostic procedure instead of the open biopsy?

DR. JAQUA: Yes, the disease can be diagnosed by needle biopsy. However, you must remember that this may be a focal process which could have been missed on a blind needle biopsy. Needle biopsies are much more sensitive indicators of diffuse disease. DR. ULRICH: I would like to re-emphasize that if one suspects drug induced allergic interstitial nephritis, a useful technique is a Wright stain of a spun urinary specimen in search of eosinophils.

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^{*} Faculty, Family and Community Medicine Program, USD Affiliated Program.

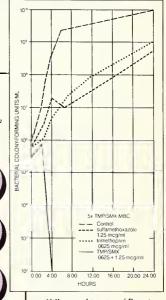
[†] Resident in Family and Community Medicine, USD Affiliated Program.



Bactericidal activity

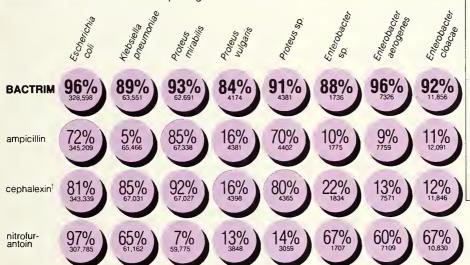
with minimal resistance

RAPID IN VITRO DESTRUCTION
OF F COLI*



Kill curve kinetics of Bactrim and its individual components against E. coli in vitro. ¹

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials



[†]Analogous to cephalothin, the primary antibiotic disc used in testing. Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82 Numbers under percentages refer to the projected number of isolates tested.

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*^{1,2} and on clinical isolates of *E. coli*, *Klebsiella-Enterobacter*, *Proteus mirabilis* and *Morganella morganii*³—the most common causative organisms of urinary tract infections. More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms. In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.

Resistance to Bactrim develops more slowly than to either of its components alone in vitro.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.⁵¹² Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a singleagent antimicrobial.

Bactrim DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. for recurrent urinary tract infections

*In vitro data do not necessarily predict clinical results.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kramer MJ. Mauriz YR, Robertson TL, Timmes MD: Morphological studies on the effect of submhibitory and inhibitory doses of sullamethoxazole-trimethoprim combination on Escherichia coli. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. 3. Spicehandler J et al: Rev Infect Dis 4:562-565, Mar-Apr 1982. 4. Stamey TA: Pathogenesis and Treatment of Uninary Tract Infections. Baltimore, Williams & Wilkins, 1980, p. 13. 5. Ronald AR: Clin Ther 3:176-189, Mar 1980. 6. Cooper J, Brumitit W, Hamilton-Miller JMT: J Antimicrob Chemother 6:231-239, 1980. 7. Gower PE, Tasker PRW: Br. Med J 1:684-686, Mar 20, 1976. 8. Cosgrove MD, Morrow JW: J Urol 111:670-672, May 1974. 9. Iravani A et al: Antimicrob Agents Chemother 19:598-604, Apr 1981. 10. Schaeffer AJ, Flynn S, Jones J J Urol 125:825-827, Jun 1981. 11. Rous SN: J Urol 125:228-229, Feb 1981. 12. BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82.

Bactrim DS (trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

which follows: Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the tollowing organisms: Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii. It is recommended that initial episodes of uncomplicated urinary tract intections be treated with a single effective antibacterial agent rather than the combination. Note: The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of Haemophilus influenze or Strengoccus preumoniae when in physician's judgment it offers.

influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of Haemophilus influenzae or Streptococcus pneumoniae when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of Shigella flexneri and Shigella sonnei

when antibacterial therapy is indicated.

Also for the treatment of documented Pneumocystis carinii pneumonitis Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernictural interactions and may be a meeting of services a

mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL

PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptooccal tonsillopharyngitis have higher incidence of bacteriologic failure when treated
with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is
much more limited but occasional interference with hematopoiesis has been reported
as well as an increased incidence of thrombopenia with purpura in eldetic patients of as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBCs are recommended: therapy should be discontinued if a significantly reduced count of any formed blood element is

noted.

Precautions: General: Use cautiously in patients with impaired renal or hepatic func-Precautions: General: Use cautiously in patients with impaired renal or hepatic function, possible folate deliciency, severe altergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinal-yses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin, reassess coagulation time when administering Bactrim to these patients. Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if notential hepetits in the fetus.

sulfamethoxazole may interfere with lolic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. Blood dyscrasas: Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. Aflergic reactions: Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, extofiative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthrafgia and allergic myocarditis. Gastrontestinal reactions: Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. CNS reactions. Headache, peripheral neuritis, mental depression convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, latique, muscle weakness and nervousness. Miscellaneous reactions: Drug lever, chills, toxic nephrosis with oliquira and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similanties to some gotirogens, diuretics (acetazolamide, thiazides) and oral hypoglycemia agents, sulfonamides have caused rare instances of gotter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may

and oral hypoglycemic agents, sulfonamides have caused rare instances of gotter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies. Dosage: Not recommended for infants less than two months of age. URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN.

ADULTS AND EXPENSIVE AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN.

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses or 10 days. Use identical daily dosage for 5 days for shigellosis. For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

below 15 mi/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or

4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per

24 hours in equal doses every 6 hours for 14 days. See complete product information
for suggested children's dosage table.

Supplied: Double Strength (DS) tablets, each containing 160 mg trimethoprim and
800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose* packages of 100;
Prescription Paks of 20. Tablets, each containing 80 mg trimethoprim and 400 mg

sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription

Paks of 40. Pediatric Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz

(1 pint). Suspension, containing 40 mg trimethoprim and 200 mg sulfamethoxazole

per tea spoonful (5 ml); truit-licorice flavored—bottles of 16 oz (1 pint).



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BRIFF SUMMARY

PROCAROIA* (nifedipine) CAPSULES

PROCARDIA '(intediprine) CAPSULES For Oral Use INDICATIONS ANO USAGE: I. Vasospastic Angina: PROCARDIA (intediprine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provided by a proportion, and a significant provided that the appropriate of angina and provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g. where pain has a variable threshold on exertion or in unstable angina where electricardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina): PROCAROIA is indicated for the management of chronic stable angina (detort-associated Angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PRDCARDIA has been effective in contributed trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available infor-mation is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When in-

patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.) CONTRAINDICATIONS: Known hypersensitivity reaction to PROCAROIA.

WARNINGS: Excessive Hypotension. Although in most patients, the hypotensive effect of PROCAROIA is modest and well tolerated. occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

blockers
Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PRDCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PRDCARDIA freated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PRDCARDIA to be washed out of the body prior to surgery.

Increased Angina. Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased disastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawai: Patients recently withdrawn from beta blockers may develop a with-

Pesuling from increased near rate alone

Beta Blocker Withdrawal. Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines Initiation of PRDCARDIA treatment will not prevent this occurrence and might be expected
to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of
increased angina in a setting of beta blocker withdrawal and PRDCARDIA initiation. It is important
to these base blockers it possible. to taper beta blockers it possible, rather than stopping them abruptly before beginning

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure affer beginning PROCAROIA. Patients with tight aortic stenosis may be at greater risk for

failure affer beginning PROCAROIA Patients with tight aortic stenosis may be at greater risk for such an event
PRECAUTIONS: General; Hypotension: Because PROCARDIA decreases peripheral vascular
resistance, careful monitoring of blood pressure during the initial administration and titration
of PROCARDIA is suggested. Close observation is especially recommended for patients already
taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema. Mild to moderate peripheral edema, typically associated with arterial vasodiation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with
PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic
therapy. With patients whose angina is complicated by congestive heart failure, care should be taken
to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.
Orig interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience
on over 1400 patients in a non-comparative clinical trail has shown that concomitant administration
of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional
literature reports suggesting that the combination may increase the likelihood of congestive heart
failure, severe hypotension or exacerbation of angina.
Long-acting intrates: PROCARDIA may be safely co-administered with nitrates, but there have
been no controlled studies to evaluate the antianginal effectiveness of this combination.
Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve
normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two
hundred patients with congestive heart failure during which digoxin blood levels were not measured. digitalis toxicity was not observed. Since there have be

Pregnancy Category C. Please see full prescribing information with reterence to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

AOVERSE REACTIONS: The most common adverse events include dizziness or light-headedness.

AOVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patents. transient hypotension in about 5%, palpitation in about 2% and syncope in about 05%. Syncopal episodes did not recur with reduction in the dose of PRDCARDIA or concomitant antianginal medication. Additionally, the following have been reported muscle cramps, nervousness dyspinea, nasal and chest congestion, diarrhea, constipation, initiammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruntus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PRDCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension. In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in however, that some or many of these events were observed. The proposition of the

Interature

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of infedipine

PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 00692600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59 to 77°F (15° to 25°C) in the manufacturer's original container.

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"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks, taking fewer nitroglycerin tablets, doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

*Procardia is indicated for the management of:

1) Confirmed vasospastic angina.

2) Angina where the clinical presentation suggests a possible

vasospastic component.

3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.



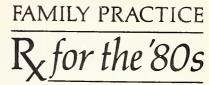
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S Chapter News





SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS 3001 South Holly Avenue Sioux Falls, SD 57105

1983 BLACK HILLS SUMMER SEMINAR

Nearly one hundred registrants from 13 states, including South Dakota Family Practice Residents, attended the 14th Annual Black Hills Summer Seminar held at the Howard Johnson Motor Lodge in Rapid City on August 11-13, 1983. They heard timely topics in Forensic Medicine, Agricultural and Occupational Medicine, and Psychiatry presented by a faculty consisting of Bradley Randall, M.D.; Thomas E. Henry, M.D.; Donald P. Morgan, M.D., Ph.D.; Kelley J. Donham, D.V.M.; Edward H. Peters, M.D.; Joseph H. Talley, M.D.; and William H. Arbes, Ph.D.

National AAFP President Gerald Gehringer and his wife Myrtle of LaPlace, Louisiana were guests of SDAFP for this meeting. Dr. Gehringer offered comments at the Board meeting and to the seminar registrants and spouses during the Thursday noon luncheon. Dr. Gehringer, specifically remarked about the important need and place for family practice in the competent continuing comprehensive delivery of health care in America, both now and in the future.

Also attending the seminar and speaking to the members assembled was Mr. Gary McMahan, Executive Vice President of the Family Health Foundation of America, the philanthropic arm of "family practice." Visiting chapter officials from Iowa, Minnesota, and Nebraska attended the seminar.

Sponsors for this meeting included Marion Laboratories, Wyeth Laboratories, South Dakota Blue Shield, Ciba Pharmaceutical Company, Geigy Pharmaceuticals, Ortho Pharmaceutical Corporation, Eli Lilly & Company, Dista Products, E. R. Squibb & Sons, Bristol Laboratories, Roche Laboratories, Searle Laboratories, Abbott Laboratories, A. H. Robins Company, Burroughs Wellcome Company, and The Upjohn Company (sponsor of Joseph Talley, M.D.).

Paul K. Aspaas, M.D. of Dell Rapids, South Dakota and Lloyd J. Sweeney, M.D. of Sun City, Arizona were recognized for their considerable efforts as co-founders of the Black Hills Summer Seminar, just completing its 14th year. Additional persons instrumental in development of this scientific program include Buck Williams, M.D. of Sioux Falls (ACOG) and Donald L. Scheller, M.D. of Arlington, South Dakota.

The Sioux Falls Family Practice Residency Program held a reunion during the seminar, commemorating the 10th anniversary since the founding of the program in May 1973. James Oakland, M.D. and his wife Carol of Sioux Falls served as Chairpersons for this event which saw 18 residency graduates, from a total of 66, return for this reunion. Honored guests included founding Director Lloyd J. Sweeney, M.D. and his wife Lois, now retired, of Sun City, Arizona. Reunion events included a Thursday afternoon social event and a Friday evening dinner and dance at the historic Alex Johnson Hotel in downtown Rapid City.

Registrants enjoyed a change in the scientific sessions format that included AM talks, allowing the afternoons and evenings for additional time to enjoy the plush greenery and scenery of the historic Black Hills, and an opportunity to attend the Central States Fair and Rodeo.

The SDAFP Education and Legislative Committees met during this seminar. The Education Committee finalized the pro-

gram for the 1984 Black Hills Winter Ski Seminar, scheduled for the Holiday Inn of the Northern Black Hills in Spearfish February 2-4, 1984. The scientific sessions will include topics on Musculo-skeletal Disorders, including Arthritis, and Pulmonary Medicine. Preliminary plans were made for the 1984 Black Hills Summer Seminar scheduled for the Howard Johnson Motor Lodge in Rapid City, August 9-11, 1984. The Legislative Committee discussed the forthcoming legislative session and the need for continued legislative support of the Sioux Falls Family Practice Residency Program. In addition, certification of midwives, as an extension of the Nurse Practitioner Act, was discussed

Officers and Delegates for 1983-84 elected and installed at this meeting were:

Officers: President: Lawrence W. Finney, M.D., Sioux Falls; President-Elect: Charles L. Swanson, M.D., Pierre; Vice President: Michael Brown, M.D., Spearfish; Vice President: Richard Finley, M.D., Rapid City; Vice President (new): Richard W. Honke, Il, M.D., Wagner; Secretary-Treasurer: L. H. Amundson, M.D., Sioux Falls; Past President: Herbert A. Saloum, M.D., Tyndall.

Delegates: Richard W. Friess, M.D., Sioux Falls; Bruce C. Lushbough, M.D., Brookings.

Alternate Delegates: Lawrence W. Finney, M.D., Sioux Falls; Herbert A. Saloum, M.D., Tyndall.

PATIENT EDUCATION TIPS

Good Health Habits Help You Beat the Odds

What do uncontrolled high blood pressure, overweight, smoking, and a high cholesterol diet have in common? They all increase the odds for getting heart disease.

How much? The answer is different for each person, depending on family history, and on how many of these "health risks" one has. The physician can explain how big a chance one may be taking with their health by not controlling risks that can be modified or eliminated.

How much risk is too much? Ask yourself. Then advise your patients in changing everyday habits to help beat the odds.



OUTREACH TO PATIENTS

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S President's Page



Six physicians laid their lives on the line when they signed the Declaration of Independence in 1776. The crime was high treason and the penalty was death. All signers served in the Continental Congress. Samuel Freeman Miller, the only physician ever appointed, served on the United States Supreme Court from 1861 to 1890. He was a lawyer, although there are no constitutional or statutory requirements for membership on the court, including a legal background. Political leadership was a hallmark of the medical profession in the eighteenth, and to a degree in the nineteenth centuries in America. It still is, in other parts of the world.

Only one physician, Dr. Robert Giebink, served in the last legislature in South Dakota. Physicians have much to offer to good government, but for most of us the sacrifice is too big.

The South Dakota Physicians Committee was established in 1960. Later the name was changed to South Dakota Political Action Committee, SoDa-PAC. Dues were first collected in 1962. The American Medical Political Action Committee, AMPAC, was established at the same time. Regular members pay \$40, and sustaining members pay \$100 per year. Half goes to SoDaPAC, and half to AMPAC. In the past five years SoDaPAC gave more than \$27,000 to 205 state candidates and over \$900 to two national

candidates. The figures are not readily available, but congressional candidates from South Dakota have received the maximum \$5,000. Our chairman this year is Dr. Nat Whitney. We needn't lay our lives on the line, but we can help Nat to help AMPAC-SoDaPAC encourage the best qualified candidates regardless of their party affiliations, and in a small way perhaps encourage political leadership.

Joseph N. Hamm, M.D., President South Dakota State Medical Association

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making body.

This Is Your Medical Association

The American Hospital Association House of Delegates has named **Theodore H. Sattler, M.D.,** Yankton, as one of 15 delegates to begin serving a three-year term on the board beginning in 1984. The House of Delegates is the AHA's highest policy

* * *

Robert R. Rietz, M.D., a native of Mitchell, has recently moved to Brookings to open his practice. He is a graduate of Creighton University School of Medicine and served on the school's faculty from 1975-1983. He did his internship and residency at the University of Nebraska Medical Center.

Dr. Rietz and his wife have four children.

* * *

Carroll Clark, M.D., Watertown, was awarded the Internist of the Year Award at a recent meeting of the combined South Dakota Chapter of the American Society of Internal Medicine and the American College of Physicians. Dr. Clark was cited for his contributions towards the specialty of Internal Medicine and for his involvement in community activities. His is only the fourth such award granted by the Chapter since its formation in 1951. Other recipients have included Dr. John Calene, Aberdeen (deceased), Dr. Don Kegaries, Rapid City (deceased), Dr. Ted Sattler, Yankton.

* * *

Tschetter-Hohm Clinic, Huron, has a new surgeon, **Dr. Fred Schneider.** He is a native of Billings, Montana and attended medical school at the University of Oregon at Portland. He served his residency at St. Francis Regional Medical Center, Wichita, KS.

Dr. Schneider and his wife have two sons.

* * *

Wagner has a new physician, Gary Bubak, M.D., internal medicine. Dr. Bubak is a native of Sisseton. He received his MD degree from the University of Minnesota Medical School after receiving his undergraduate degree from the University of South Dakota. He completed a three year residency at the Tulane University School of Medicine in New Orleans, LA.

Dr. Bubak's wife Nancy will serve as his clinic nurse.

Three new physicians, all South Dakota natives and graduates of the University of South Dakota Medical School, have joined the staff of the Brown Clinic in Watertown.

Dr. Marlin Lamb received his medical degree in 1980 and served his internship and family practice residency at St. Luke's Regional Medical Center and Marion Health Center in Sioux City, Iowa. Dr. and Mrs. Lamb have four children.

Dr. Catherine Gerrish graduated from USD in 1978 and served her internal medicine residency at University Hospitals, Case Western Reserve Uni-

versity, Cleveland, Ohio.

Dr. Edwin Gerrish received his medical degree in 1978 and served his general surgery residency at University Hospitals, Case Western Reserve University also.

Drs. Catherine and Edwin Gerrish are husband and wife and have one son.

Dr. Russell Orr, Sioux Falls, has been elected vice chairman of the South Dakota Section of The American College of Obstetricians and Gynecologists for a three-year term. Dr. Orr is a clinical association professor at the University of South Dakota School of Medicine and is a private practitioner in Sioux Falls.

Warren N. Golliher, M.D., Spearfish, has completed continuing education requirements to retain active membership in the American Academy of Family Physicians.

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Dr. Robert E. Nelson, Sioux Falls, was elected president and **Dr. Dale Berkebile,** Rapid City, was elected secretary of the South Dakota chapter of the American College of Surgeons at the chapter's recent annual meeting.

* * *

Edward Wegner, M.D., Watertown, recently joined the staff of the Medical Arts Clinic in family practice. Dr. Wegner was born in Sioux City, Iowa and was raised in Sioux Falls. He received his BA from Augustana College in 1976 and his MD from the University of South Dakota Medical School in 1980. He served his internship and residency at Tulsa Medical College, University of Oklahoma.

* * *

Dr. Jerome Bentz has recently opened his practice in Platte. Dr. Bentz, a native of South Dakota, received his medical education at the University of South Dakota Medical School and recently completed a family practice residency in Cedar Rapids, Iowa.

He and his wife have two children.

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S Medicine

Pemphigus: A Review of Current Concepts

Kim M. Lorenzen, M.D.*

ABSTRACT

Pemphigus is a rare, autoimmune blistering disease involving the skin and mucous membranes. Although there are four distinct variants of the disease, histopathologically, each is characterized by the presence of intraepidermal acantholysis. This disturbance in the normal cell-tocell adhesion produces the typical vesicles or bullae seen in pemphigus. Immunofluorescent studies have demonstrated the presence of IgG antibodies in the intercellular substance of the

Pemphigus describes a group of rare, chronic blistering diseases of unknown etiology. The disease is found throughout the world and it has an unexplainably higher incidence in the Jewish race. Pemphigus is a disease that affects persons in middle and old age. The mean age of onset is the sixth decade, the range being 12 to 88 years old. The disease appears to affect men and women equally.

Pemphigus has four variants that are clinically, histologically and immunopathologically distinct. Lever has recently classified pemphigus into two categories: pemphigus vulgaris and pemphigus foliaceous. Pemphigus vegetans is considered to be a variant of pemphigus vulgaris, and pemphigus erythematosus is a variant of pemphigus foliaceous.

PEMPHIGUS VULGARIS

About 80% of all pemphigus patients seen have pemphigus vulgaris. About 60% of patients present with oral lesions initially. Mucous membranes of the pharynx, esophagus, conjunctiva, larynx, urethra,

epidermis and in the serum. An interesting relationship has been observed between pemphigus, thymoma and myasthenia gravis. Pemphigus has been associated with an increased occurrence of malignancy. In addition, possible associations with several other diseases have been noted. In the precorticosteroid era, pemphigus was a fatal disease. However, today, steroids, as well as immunosuppressive agents, gold, sulfones and antimalarial drugs have all been used alone or in various combinations to greatly improve the prognosis.

cervix and rectum may also be involved. Generally, five months elapse between the onset of oral lesions and dissemination to the glabrous skin. Pemphigus involving only the glabrous skin occurs in about 10% to 15% of patients.²

Nikolsky's sign is invariably positive in pemphigus.

The primary lesions are clear, tense vesicles or bullae of various sizes that usually arise from normal, uninflamed skin although they may be atop an erythematous plaque or urticarial base. They commonly first affect the head, trunk and mucosal surfaces of the body orifices. Within two or three days, the vesicle contents become turbid and the lesions become more flaccid; they soon rupture, producing painful, raw denuded areas. Scaling and crusting are common.

Nikolsky's sign is invariably positive in pemphigus. An ancillary sign is produced when pressure is applied over the greater portion of the surface of a recently developed blister and the blister enlarges peripherally by extravasation of the fluid into the

^{*} First year pathology resident, U. of Nebraska, Omaha, NE. Formerly fourth year medical student, USD School of Medicine, Sioux Falls, SD at the time of this article.

layers of the epidermis.³

Blisters in the oral cavity are usually multiple, irregular, painful, persistent, and superficial. The entire oral cavity may be affected, but involvement of the gingiva, palate, and buccal mucosa is seen most frequently. It is rare to see pemphigus in the oral cavity without subsequent skin involvement.²

The earliest histopathologic changes in pemphigus vulgaris are edema and disappearance of the epidermal intercellular bridges in the lower layer of the stratum germinativum.

Although localized involvement of one area of the glabrous skin or the mucous membranes may persist for weeks or months, gradually more lesions develop on the skin. With the onset of new lesions, the patient may experience some pruritis, burning and local discomfort, but pain becomes a predominant symptom once the bullae rupture. An offensive, nauseating odor emanates from the bullae. Because of the loss of protein in the blister fluid, along with the lack of repletion of body stores and the poor nutritional status due to the oral involvement, the untreated patient suffers from progressive malnutrition and debility,³ with death occurring within an average of eighteen months.

The earliest histopathologic changes in pemphigus vulgaris are edema and disappearance of the epidermal intercellular bridges in the lower layer of the stratum germinativum (Malpighi). The loss of epidermal cell-to-cell adhesion (acantholysis) produces intraepidermal clefts and bullae.² Suprabasal acantholytic vesicles are diagnostic of pemphigus vulgaris.³ The basal cell layer in pemphigus vulgaris shows the so-called tombstone-row arrangement, whereby the basal cells show separation from the overlying epidermal cells and each other, while the structures connecting the basal cells to the dermis remain intact.⁴

Little inflammatory reaction accompanies the acantholytic process. A few eosinophils may be seen within the bulla and in the dermis beneath such a lesion. Some early lesions show spongiosis of the epidermis with a diffuse infiltration with eosinophils, referred to as eosinophilic spongiosis.³ Spongiosis is defined as intercellular edema that widens the intercellular space and stretches the intercellular bridges, imparting a sponge-like appearance upon the epidermis.⁵ Although eosinophilic spongiosis is relatively rare, its recognition as an early manifestation of pemphigus vulgaris, as well as pemphigus foliaceus, is important since it may occur in biopsy sections that do not show any evidence of acantholysis. However, as shown by Knight et al,

eosinophilic spongiosis can be seen in other bullous eruptions and thus is not diagnostic of pemphigus.⁴ In addition, at times a transition between eosinophilic spongiosis and acantholysis can be seen.³

Electron microscopy studies by Hashimoto and Lever have shown that wherever acantholysis is beginning, the intercellular cement substance, or glycocalyx, is partially or entirely dissolved. Thus, the current belief is that the cement substance dissolves first in the nondesmosomal areas then in the desmosomal areas and at the intermediate junction areas later. The dissolution of the cement substance around cells in the stratum malpighian progresses to the development of acantholytic cells. The connection between the basal cells and dermis does not contain any intercellular cement substance; therefore, it remains intact, accounting for the tombstonerow appearance eluded to earlier.

For the purposes of diagnosis, direct immunofluorescence of uninvolved skin is the most sensitive and accurate test that can be used.

Immunoelectron microscopy studies have used immunoperoxidase-labeled antihuman IgG to locate in vivo bound IgG in pemphigus vulgaris. In the uninvolved skin, the reaction product is found in the intercellular spaces. Within areas of acantholysis, the widened intercellular spaces do not show any reaction product but the electron-dense material is seen deposited on the surface of the epidermal cells in a discontinuous globular pattern.²

Immunologic studies have shown that for the purposes of diagnosis, direct immunofluorescence of uninvolved skin is the most sensitive and accurate test that can be used. Indirect immunofluorescence testing for antibody in the serum is an inferior diagnostic measure because it may be negative in early cases. Essentially all patients with pemphigus have IgG antibodies in the intercellular areas of the epidermis as demonstrated by direct immunofluorescence. In addition, 70% to 85% of patients have IgG antibody in their sera that will react with normal skin (indirect immunofluorescence).

The antigen inducing these antibodies is unknown. It is believed to probably be a specific carbohydrate moiety in the glycocalyx of normal human epidermis, since antibodies bind to skin from normal people as well as to those with pemphigus. The glycocalyx is a coating over the membrane of epidermal cells that acts as an intercellular cement. Although Nabai and Rahbari have reported multinucleated epithelial cells in association with pemphigus vulgaris, they are unsure as to whether this is

merely an occasional finding in the disease or whether the herpetic element represents the triggering factor of the disease or an opportunistic infection. Otherwise, there is no real evidence that pemphigus antigen is of viral origin and it is not an HLA antigen or blood group substance.

Intercellular antibodies may be involved in the pathogenesis of pemphigus. The antibodies usually represent all subclasses of IgG. They apparently are capable of complement fixation, although complement may not be necessary for induction of pemphigus. It is uncommon to find complement in the intercellular areas of uninvolved skin, despite the presence of IgG antibody. In lesional skin, C3, C3 proactivator, properdin and C1q have all been seen in the intercellular areas and imply local activation of both classical and alternative pathways. Total hemolytic levels of complement in blister fluids are low, which also implies consumption or fixation of complement in lesional skin. However, since "heatinactivated" pemphigus sera devoid of complementfixing ability will still induce acantholysis in tissue explants, consumption of complement in pemphigus may only be a secondary phenomenon unrelated to blister formation.

A familial incidence has been observed in pemphigus vulgaris, as well as in the other three variants of pemphigus.

In 1971, Sams and Jordon were unable to produce disease or lesions by transfusing human pemphigus plasma into monkeys, despite the strong binding of the antibody to the epidermal intercellular space. Significant blister formation did occur however, in animals sensitized to monkey esophagus if the sensitized rabbits were first subjected to an epidermal insult such as a chemical irritant or physical trauma.²

In 1978, Farb et al found that the addition of pemphigus serum to a monolayer culture of mouse epidermal cells resulted in antibody binding and reduced cellular adherence to the plastic culture dish. The process was prevented by serum protease inhibitors. Similar results were obtained by Singer et al about two years later. These data suggest that pemphigus antibody appears to induce epidermal cells to activate cellular proteinases, which then degrade the glycocalyx causing cell dyshesion and acantholysis. 10

A familial incidence has been observed in pemphigus vulgaris, as well as in the other three variants of pemphigus. Although in many of the earlier studies, immunofluorescent or histologic evidence was lacking, Ahmed and Sofen studied two different families whose members had clinical, histologic and

immunopathologically proven pemphigus vulgaris. 11

Evidence that genetic factors may play a role in the susceptibility to pemphigus comes from recent studies indicating a strong linkage between pemphigus and HLA-DRW4. ¹² Earlier studies have demonstrated a linkage of HLA-A10 and HLA-A13 in patients with pemphigus. Further studies in affected families are necessary to help understand the interaction between genetic and environmental factors in the pathogenesis of pemphigus vulgaris. ¹¹

The Tzanck test gives laboratory confirmation of the presence of the acantholytic process in pemphigus vulgaris. The hemogram is of some significance prognostically. Increasingly severe anemia suggests that the pemphigus is a malignant type with a poor prognosis; leukocytosis increases with the severity of the disease. Eosinophilia may be present but it decreases with the progression of the disease. In advanced disease, serum protein electrophoretic patterns show decreased albumin and increased α_1 -globulin and α_2 -globulin. Serum sodium, chloride and calcium decrease while potassium increases as the disease progresses.³

PEMPHIGUS VEGETANS

Pemphigus vegetans is considered to be a phase of pemphigus vulgaris in which large, verrucous and granulomatous plaques and vegetating masses, rather than blistering, are the most prominent features of the disease. Pemphigus vegetans has been divided into two varieties depending on the types of primary lesions from which the vegetations develop and its clinical course. ¹³

Pemphigus vegetans, the Neumann type, usually behaves like pemphigus vulgaris except that it appears at an earlier age. Blisters are the primary lesions and most of the resulting denuded areas, especially of the face, axillae, genitals and other intertriginous areas, develop vegetations that eventually become verrucoid. Oral lesions are almost always present. ¹ Its prognosis is similar to pemphigus vulgaris. ²

Pemphigus vegetans, the Hallopeau type, is a benign form of pemphigus that has a chronic, prolonged course. The primary lesions are pustules that coalesce, extend peripherally and become vegetative, with a preference being noted in the axillae, perineum and other flexural areas.¹

Histologically, the initial lesion has the same appearance as pemphigus vulgaris but later there is marked acanthosis of the epidermis, resulting from the downgrowth, and an increase in papillomatosis. In addition, abscesses composed almost entirely of eosinophils develop within the epidermis. A sparse dermal inflammatory infiltrate of eosinophils and

plasma cells is usually present. As the lesions become more established, the abscesses disappear, and marked hyperkeratosis develops over the hyperplastic epidermis, and the histologic pattern becomes penspecific.

Indirect immunofluorescence studies have shown antibodies against epithelial intercellular substance in patients with pemphigus vegetans. Electron microscopy has shown a reduction in the number of desmosomes in affected regions; also, degenerative changes, such as disappearance of cytoplasmic organelles and vacuole formation, are common in cells close to bullae. The hemogram and serum electrolyte levels are more nearly normal than in pemphigus vulgaris. A marked increase in serum γ -globulin has been reported. ¹³

PEMPHIGUS FOLIACEUS

Pemphigus foliaceus, which presents as recurrent crops of superficial blisters with considerable crusting and scaling, usually pursues a benign, prolonged course although complete recovery may occur. The disease most commonly affects middle-aged persons, however, it is almost equally common in males and females. Emotional upset, nervous strain and fatigue, and psychic shock exacerbate the skin lesions in about one-third of the patients. Less frequently, menstruation and excessive exposure to increased heat and sunlight are implicated.

A definite histologic diagnosis of pemphigus foliaceus requires finding acantholytic epidermal cells in bullae situated high in the epidermis, beneath the stratum corneum, within the granular layer, or in the uppermost part of the rete Malpighi.

Pemphigus foliaceus occurs in two forms — generalized, presenting as a generalized exfoliative dermatitis and localized, with scattered lesions present primarily over the scalp, butterfly area of the face, and upper part of the trunk. The patient usually describes the initial lesion as a scaling papule or plaque, and less frequently as a vesicle or bulla, on the face, neck, or upper trunk. The vesicles and bullae are flaccid, very superficial and situated on either normal skin or an urticarial base that ruptures very easily. As a result, scaling, oozing and crusting, with a cornflake-like appearance, are prominent. Crusting takes place centrally while at the same time the bullous component of the lesion is extending peripherally. ¹⁴

Lesions usually begin on the face and later spread. Oral lesions are generally not present.² Most often the patient complains of local discomfort, described

as burning, itching, pain or a dry, pulling sensation; with generalized involvement marked discomfort of the skin occurs. The skin lesions have a characteristic musty smell but lack the nauseating odor emanating from lesions in pemphigus vulgaris.

A definite histologic diagnosis of pemphigus foliaceus requires finding acantholytic epidermal cells in bullae situated high in the epidermis, beneath the stratum corneum, within the granular layer, or in the uppermost part of the rete Malpighi. Only rarely seen, but perhaps representing the earliest type of histologic change, is the presence of polymorphonuclear leukocytes, predominately eosinophils, in small nests high within the epidermis, that may not be associated with any acantholysis of epidermal cells.

Pemphigus erythematosus is generally considered to be a localized form of pemphigus foliaceus.

It is important to obtain a biopsy specimen from an early lesion because with aging of the lesions, acantholytic cells extend deeper into the epidermis and confusion with pemphigus vulgaris may occur. As in pemphigus vulgaris, intercellular epidermal antibodies can be demonstrated in pemphigus foliaceus; however, in the latter, they often appear to occupy a position high in the epidermis. Perhaps as many as one-third of patients demonstrate greater fluorescence of antibodies to intercellular substance in the upper, as opposed to the lower, epidermis. Because the patient's general health is not adversely affected, except in those with extensive and prolonged cutaneous involvement, routine laboratory evaluation does not reflect any evidence of the disease. At most, a minimal peripheral eosinophilia may be seen. 14

PEMPHIGUS ERYTHEMATODES

Pemphigus erythematodes, also known as pemphigus erythematosus or Senear-Usher syndrome, is characterized by erythematous, scaling, hyperkeratotic plaques frequently seen in a butterfly distribution over the nose and malar areas, as well as combinations of bullae, raw denuded areas and crusted lesions on the trunk. There is usually no visceral involvement.² Pemphigus erythematosus is generally considered to be a localized form of pemphigus foliaceus because of the similar histologic appearance, the frequent presence of few lesions for months in both, the absence of oral lesions, the good health of patients, and the better prognosis than in other types of pemphigus.¹

Senear and Usher described a group of eleven

patients whose lesions appeared mainly over the scalp, face and upper part of the trunk, particularly the sternal and interscapular areas. Intertriginous areas were sometimes involved, but lesions rarely if ever occurred on the extremities. On the trunk, bullae had a wrinkled, flaccid appearance with a thin roof that ruptured easily to form raw denuded areas, some of which healed with hyperpigmentation. At other times, the bullae ruptured and lesions resembling keratoses, psoriasis and severe impetigo developed. Symptoms were of little consequence with most patients being either asymptomatic or having only mild pruritis.

Thymoma and myasthenia gravis may be seen in association with pemphigus.

Biopsy material taken from patients diagnosed clinically as having pemphigus erythematosus shows the same histologic pattern as that seen in the generalized forms of pemphigus foliaceus. Some patients with pemphigus erythematodes have, in addition to pemphigus antibodies, antinuclear antibodies and bound IgG in the basement membrane area. Positive LE cell preparations have also been reported.¹⁵

CLINICAL ASSOCIATIONS

Thymoma and myasthenia gravis may be seen in association with pemphigus. The triad of myasthenia gravis, thymoma, and pemphigus is well-documented, with pemphigus foliaceus or erythematosus occurring more frequently than pemphigus vulgaris. In most patients, myasthenia gravis has preceded the development of pemphigus. About 50% of patients with myasthenia gravis and pemphigus have thymomas. In most of these patients, pemphigus is usually found to develop after thymectomy. It has been shown that 15% to 45% of patients with myasthenia gravis and thymoma, but with no clinical skin disease, have intercellular substance antibody detectable in their sera. In these patients, the clinical appearance of pemphigus at a subsequent time is believed to be triggered by a mechanism such as thymectomy, irradiation, sunlight or drugs. In addition, pemphigus with thymoma but without myasthenia gravis has been reported in a patient with rheumatoid arthritis, lupus erythematosus and in patients with systemic lupus erythematosus and thymoma.2

A relationship appears to exist between pemphigus erythematosus and systemic lupus erythematosus. Evidence for this comes from several studies, with one in particular showing that 31% of patients had a positive antinuclear antibody titer,

The occurence of malignancy is greater in patients with pemphigus than in age and sexmatched normal persons.

81% a positive lupus band test (deposits of immunoglobulin or complement, or both, at the dermoepidermal junction) on exposed skin, and 23% a positive lupus band on unexposed skin. However, although the lesions of pemphigus erythematosus may resemble systemic lupus erythematosus clinically, they differ histologically.

The occurrence of malignancy is greater in patients with pemphigus than in age- and sex-matched normal persons. Krain found that 54% of patients with pemphigus who developed a neoplasm had a malignancy of the lymphoid or reticuloendothelial system.² However, the type of malignancies varied and included carcinomas of the ovary, stomach, breast, uterus, bladder, esophagus, bronchus and skin, as well as Kaposi's sarcoma and lymphoma. Of these patients, 75% had pemphigus vulgaris; 25% had pemphigus foliaceus.³

In 1969, Degos et al described a bullous dermatosis resembling pemphigus in a patient with Wilson's disease who had been treated with penicillamine for a period of 4 years. Subsequently, thirty additional cases of so-called penicillamine-associated pemphigus have been reported, the majority in patients with rheumatoid arthritis receiving D-penicillamine. ¹⁶

The great majority of cases of penicillamine-associated pemphigus were pemphigus foliaceus. Most cases of pemphigus started between six and twelve months after initiation of penicillamine therapy. In most instances, the pemphigus was fairly mild and subsided spontaneously shortly after penicillamine was discontinued. Although it is not known how penicillamine may induce pemphigus, it can be assumed that penicillamine, possibly by the action of its sulfhydryl groups, alters the intercellular cement substance into an antigenic structure with subsequent antibody formation. 4

The bullous dermatosis associated with penicillamine has been considered a form of pemphigus because of histologic and immunologic similarities. Histologic examination of the penicillamine-associated lesions has consistently demonstrated intraepidermal acantholysis. Direct immunofluorescence has detected intercellular deposition of immunoglobulin in the skin in all cases in which it has been used. Indirect immunofluorescence used to detect circulating serum antibody directed against the intercellular substance has been positive in 72% of those cases tested.

More recently, however, Troy et al questioned whether penicillamine-associated pemphigus really

was pemphigus. They described a case in which a woman with rapidly progressive rheumatoid arthritis developed clinical features similar to those reported in previous cases of penicillamine-associated bullous eruptions, after nine months of D-penicillamine therapy. In their studies, skin lesions showed intraepidermal bullae with acantholytic cells, but the dermal infiltrate similar to cases described previously, was unusual for pemphigus. In addition, immunofluorescent findings were those of bullous pemphigoid rather than pemphigus — dermoepidermal junction deposition of immunoglobulin with circulating serum antibody directed against the basement zone. ¹⁶

TREATMENT AND PROGNOSIS

Simply stated, treatment of pemphigus generally consists of high doses of corticosteroids initially, followed by a gradual reduction in dosage once suppression of the eruption is achieved. More recently, immunosuppressants, namely methotrexate, azathioprine and cyclophosphamide, have been employed both alone and in combination with corticosteroids.3However, the "combined treatment," in which an immunosuppressant is added after the high-dosage treatment with prednisone is completed, appears superior to treatment with an immunosuppressant alone.⁴ In addition, gold therapy has been used in conjunction with corticosteroids as a corticosteroid-sparing drug, as well as alone, with good results. Sulfones have been used in the treatment of pemphigus vulgaris when the above modalities proved ineffective.³ Some patients with pemphigus foliaceus respond to the antimalarial drugs chloroquine, hydroxychloroquine or combinations thereof. 14 Sometimes antibiotics are given to reduce the risk of secondary bacterial infection. ¹³

In pemphigus, infection is the commonest cause of death, and the commonest infection is <u>Staphylococcus aureus</u> bacteremia.

In 1966, Chorzelski et al published an extensive study on the clinical importance of pemphigus antibodies. They found that serial antibody test titers decreased as the patients responded to therapy. As the titer rose, the disease frequently exacerbated. These findings emphasized the value of serial serologic studies in patient treatment. ¹⁷ In 1967, Peck et al also reported a definite relationship between clinical activity and antibody titer. ⁹ Similar findings were again found in 1978 by O'Loughlin et al. ¹⁸ and Feuerman et al. ¹⁹

However, in a 1980 article, Fitzpatrick and Newcomer stated that serial titers were not consistent enough to be used reliably as a guide to therapy or prognosis. ²⁰ In their study, there was a relationship between the change in titer and disease activity, but the timing was such that the change in titer usually occurred after the change in disease activity and was rarely predictive. Although there is general agreement that the antibody titer is elevated during active disease phases and lowered during remission, the usefulness of serial titers in patient treatment remains debatable.

Mortality from pemphigus was essentially 100% before corticosteroid therapy was available. Patients with pemphigus vulgaris died within an average of 18 months, whereas patients with pemphigus foliaceus had a more prolonged course, surviving five years. At present, the mortality for pemphigus averages about 10%. Age, duration and severity of disease all appear to be important in evaluating mortality. Mortality is higher in persons who develop the disease after age 50. More than 50% of pemphigus patients who die do so during the first year and, in addition, patients with severe disease requiring large doses of steroids have a mortality rate of up to 75%. In pemphigus, infection is the commonest cause of death, and the commonest infection is Staphylococcus aureus bacteremia.2

In summary, pemphigus describes a group of chronic blistering diseases of unknown etiology, characterized clinically by the involvement usually of healthy-appearing skin and mucous membranes, histologically by the presence of acantholysis with resultant intradermal bullae, and immunologically by the presence of circulating autoantibodies to an intercellular epidermal antigen. Although alot of progress has been made in our understanding of this fascinating disease process, further studies are necessary to answer the many questions that remain unanswered.

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S Auxiliary News

CHILD ABUSE PREVENTION

In December 1982, the AMA House of Delegates approved a plan in which the AMA, in cooperation with state medical and specialty societies and the AMA Auxiliary, will take a leadership role in a renewed attack on the widespread problem of child abuse, including sexual abuse.

At the annual meeting in June, the AMA Auxiliary launched a nationwide program to involve all auxiliaries in the effort in three major areas:

- —Parenting Education, including workshops and seminars in the community, as well as efforts to have parenting included in school health curriculum;
- —Helpful Visitor Programs directed to helping relieve stress of mothers who are considered at high risk for abuse;
- —Coalition efforts with other community organizations to promote awareness and disseminate information on a community-wide basis.

The areas chosen for AMA Auxiliary involvement in the AMA program are important areas for volunteers. Within these areas is potential for many kinds of programs: outreach to help prevent child abuse by providing outlets for parents who could become abusive; self-help groups to give parents a forum for discussion to relieve the tensions that can lead to abuse; hotlines to relieve stress at the point of crisis; speakers bureaus to help educate people on parenting, as well as how to prevent child abuse.

Many ideas for programs are available in the AMA Auxiliary Project Bank — programs such as the South Carolina statewide conference on child abuse; Parents Anonymous groups and crisis hot-



lines; pamphlets on child abuse for physicians; training volunteers to speak to local groups; or setting up crisis centers to provide emergency shelter, counseling, and intervention.

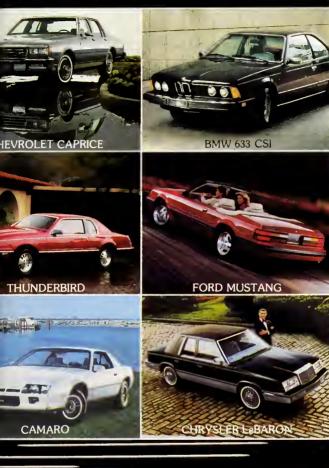
The problem of child abuse and neglect will not go away. The help of concerned people is needed if children are to be helped.

Members of the SDSMA Auxiliary are urged to become actively involved in programs dealing with the prevention of child abuse and neglect.

Marie Howland

Marie Hovland, President South Dakota State Medical Auxiliary

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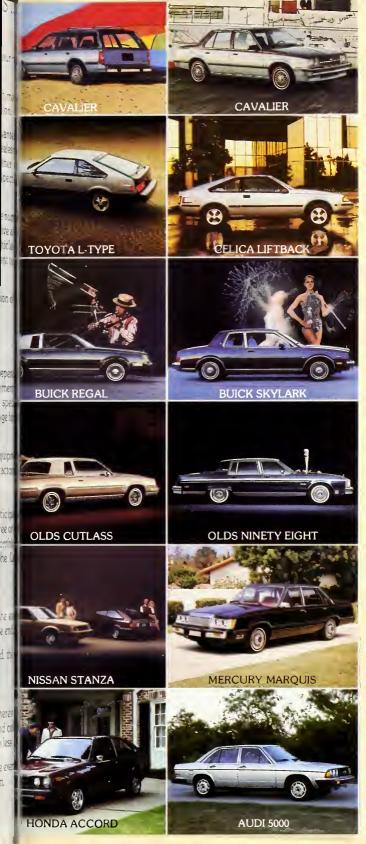
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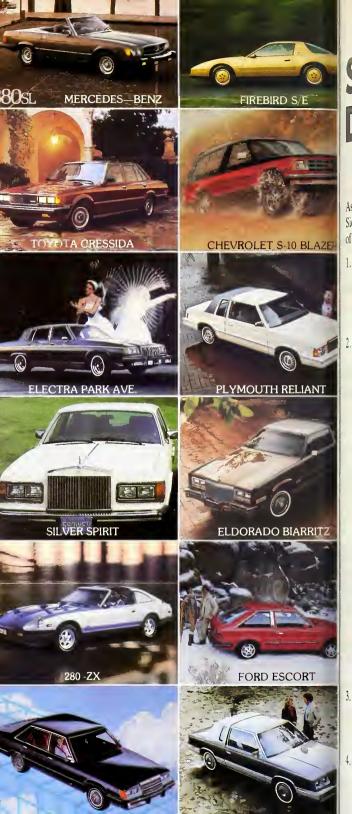
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*Many Lessors offer a purchase-option which may be exerc by the lessee during or at the end of the lease term.





S Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday, September 23, 1983, in Sioux Falls, South Dakota. Following are the items of business transacted at this meeting.

- 1. DISABILITY INCOME INSURANCE. The Council approved the disability income insurance proposal for primary or supplemental coverage for SDSMA members which was submitted by Williams Insurance Company.
- 2. PRACTICE PARAMETERS FOR NURSE MIDWIVES. The Council endorsed the position of the AAFP which states, "the use of midwives is not in the best interests of quality patient care and opposes licensure of midwives. The AAFP does not believe that the midwife can adequately substitute for the physician in obstetrics. The AAFP cannot endorse a position statement which includes advocacy of midwifery. AAFP policy has recommended abolishment of midwifery for many years while recommending production of sufficient competently trained family physicians to provide quality obstetrical services. Any trend from competently trained licensed physicians performing quality obstetrics back to midwifery must be considered a regressive step in the delivery of obstetrical services." The Council endorsed the position that if abolishment of the nurse midwife law is not possible, the Medical Association should then take action to amend the law to require on-site supervision of nurse midwives.
- 3. CPR TRAINING FOR HIGH SCHOOL STU-DENTS. The Council endorsed the concept of requiring basic cardiac life support in the high school curricula in South Dakota.
- 4. 1984 ANNUAL MEETING PROGRAM. The Council recommended that the annual meeting have both a scientific program and a risk management program, and that the risk management program meet the criteria of the St. Paul Companies so that those in attendance will be eligible for a 10 percent premium discount.
- 5. SDSMA BYLAW AMENDMENT. Bylaw amendments will be submitted to the House of Delegates in June 1984 which will limit the terms physicians can serve on Association commissions to three consecutive three year terms or a total of nine years.

- COST CONTAINMENT REPORT. This report
 was referred back to the Commission on Medical
 Service for refinement and for prioritizing recommendations for further consideration by the
 Council.
- 7. NURSERY SUPERVISORY COMMITTEE REPORT. The Council acknowledged the tone of this document and the attempt to define a high level of medical care but took action not to accept the report as written.
- 8. CO-PAYS FOR MEDICAID. The Council endorsed the proposed change from a \$1 co-pay for each service provided in a physician's office to a \$3 charge for each physician encounter.



Future Meetings

December

- Frontiers in Medicine, St. Joseph's Hosp., St. Paul, MN, Dec. 3. 7 Hrs. Category 1 credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, Dec. 7. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Coronary Heart Disease: A Comprehensive Review of Principles and Practice, St. Paul-Ramsey Med. Ctr., St. Paul, MN, Dec. 7-10. 18-62 hrs. Category I credits. Contact: Charles Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

January

- Sports Medicine Symposium, U. of Iowa, Iowa City, IA, Jan. 5-7. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- 2nd Biannual Gastrointestinal & Hepatic Diseases Conference, Mauna Kea Beach Hotel, Hawaii, Jan. 16-20. 20 hrs. Category I credits. Contact: Yvonne Brewer, M.P.H., Ed. Dir., Honolulu Med. Group Res. & Ed. Found., 550 S. Beretania St., Honolulu, HI 96813. Phone: 808-537-2211; ext. 751.
- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, Jan. 19. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- 8th Annual Hawaii Echocardiography Conference, Kahala Hilton Hotel, Honolulu, Jan. 23-27. 20 hrs. Category I credits. Contact: Yvonne Brewer, M.P.H., Ed. Dir., Honolulu Med. Group Res. & Ed. Found., 550 S. Beretania St., Honolulu, H1 96813. Phone: 808-537-2211, ext. 751.

February

- **Ophthalmology Clinical Conference**, U. of Iowa, Iowa City, IA, Feb. 1. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Cardiac Dilemmas, U. of Iowa, Iowa City, IA, Feb. 15-17. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Fifth National Cancer Communications Conference, Washington, DC, Feb. 15-17. Contact: Nancy McCormick-Pickett, Spec. Ass't., Office of Cancer Communications, Bldg. 31, Rm. 4B39, Bethesda, MD 20205.
- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, Feb. 16. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, 1A 52242.

- Current Concepts in Perinatal Medicine, Radisson Plaza Hotel, St. Paul, MN, Feb. 16-17. AMA Category I credits. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Consensus Development Conference on Analgesic Associated Kidney Disease, Masur Aud., Nat. Inst. of Health, Bethesda, MD, Feb. 26-29. Contact: Michele Dillon, Prosp. Assoc., Ste. 401, 2115 E. Jefferson St., Rockville, MD 20852. Phone: (301) 468-6555.

March

St. Moritz 1984: Advances in Diagnostic Imaging, Palace Hotel, St. Moritz, Switzerland, Mar. 24-Apr. 1. 20 hrs. Category I credits. Contact: Educational Symposia, P. O. Box 17241, Tampa, FL 33682. Phone: (813) 971-6000, ext. I112.

April

International Breast Cancer Conference, Tianjin, China, Apr. 24-27. Contact: Mr. C. H. Tu, Manager Dir., Inter-World Exchange Service, 401/2 & 404/5 Metropole Bldg., 57 Peking Rd., Kowloon, Hong Kong, China.

Minnesota Medical Association Resource Group on **Rheumatic Diseases**

Presents

RHEUMATOLOGY SEMINAR V March 6-March 13, 1984

LOCATION: Paradise Grand Hotel, Nassau, BAHAMAS

DATES: Departure from Twin Cities Airport on Tues-

day, March 6

Return to Twin Cities on Tuesday, March 13 Educational Program — March 7-11

FFF:

\$285 (educational program) Approximately: \$1378 per physician/\$324 per accompanying spouse or child (includes round-trip flight, ground transportation and accommodations for seven nights)

AUDIENCE: Primary care physicians and physicians who are involved in the care of arthritic patients.

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Common rheumatologic problems, diagno-CONTENT: sis, treatment and the course of the disease

HOURS: 20 hours, Category I/Prescribed

CONTACT: Department of CME and Meeting Services, Minnesota Medical Association, Suite 400, 2221 University Avenue SE, Minneapolis,

Minnesota 55414, 612/378-1875.

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Volume XXXVI December 1983 Number 12

SOUTH DAKOTA JOURNAL OF

Medicine

Clinicopathological Conference Twenty-Three Year Old Caucasian Female at 23 Weeks Gestation With Vaginal Bleeding and Abnormal Ultrasound Study

Epidural and Intrathecal Narcotics for Pain Relief

Table of Contents: page 3

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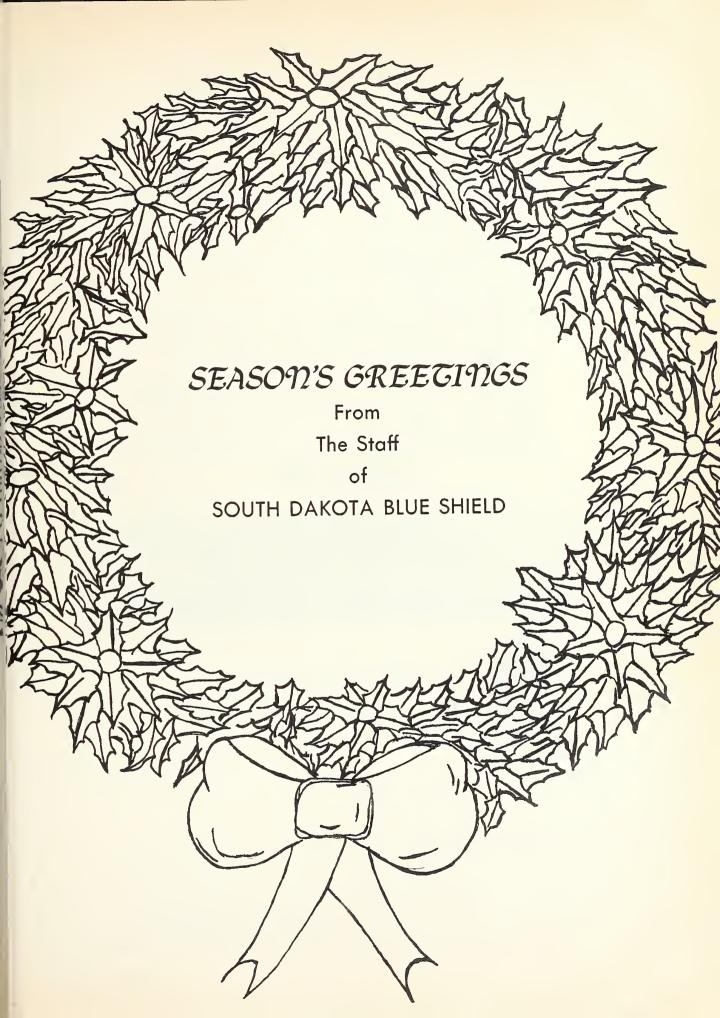
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NEXT MONTH

Clinicopathological Conference Thirty Year Old Caucasian Male With Acute Onset of Abdominal Pain

Atrioventricular Block — A Review



Best Wishes For the Kolidays and for Kealth and Kappiness Throughout the Year



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Bob Johnson, Patty Butler, Jan Anderson Jeri Spars, Dee Knobel, Susan Best

SOUTH DAKOTA STATE MEDICAL ASSOCIATION

S Clinicopathologic Conference

Twenty-Three Year Old Caucasian Female at 23 Weeks Gestation With Vaginal Bleeding and Abnormal Ultrasound Study

Kim A. Pederson, M.D.* Nancy W. Luechtefeld, M.D.† Discussers

John F. Barlow, M.D.‡ Editor

Case 920-225

This 23-year old married caucasian female entered Sioux Valley Hospital at approximately 23 weeks gestation for vaginal bleeding.

She was a gravida 2, para 0. The previous pregnancy had been a spontaneous abortion one year previously complicated by pelvic inflammatory disease requiring two episodes of dilatation and curettage and laparoscopy as well as treatment with intravenous antibiotics. Her menstrual history had otherwise been regular with menses every 30 days and a duration of about 7 days with fairly heavy flow. During the present pregnancy, the patient had not felt fetal motion and no fetal heart tones had been heard. At 17 weeks of gestation, she had had dark red vaginal bleeding and four weeks prior to admission had had bright red vaginal spotting for which she had been admitted to an outside hospital. An ultrasonic examination was read as markedly abnormal. There had been no significant other symptoms. The hemoglobin was 12.1 gm/dl. She was referred for further evaluation.

Past medical history revealed no other serious hospitalizations or illnesses. Review of systems was essentially negative. PHYSICAL EXAMINATION: Height 5'7", weight 162½ lbs., temperature 37°C, pulse 60/min and regular; respiration 20/min and regular; blood pressure 110 systolic and 60

diastolic. The patient was in no acute distress. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The breast had no masses. The heart was within normal limits of size with a regular rhythm and no murmurs. Peripheral pulses were unremarkable. Examination of the abdomen showed a uterus 22cm in fundal height. No other organs, masses, spasm or tenderness were felt. Fetal heart tones were not heard. The cervix was closed and long. The uterus was about 22 to 23 weeks gestational size on pelvic examination. There was no edema of the extremities and neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis, orange, cloudy, specific gravity 1.027; pH 6.0; negative for glucose, protein, ketones, bilirubin, moderate amount of hemoglobin; sediment 1-3 red cells/hpf. Hemoglobin 13.0 gm/dl, hematocrit 37 vol/dl, normal red cell indices, total leukocyte count $6,900/\text{mm}^3$ (6.9 x $10^9/\text{L}$) with a differential of 62% segmented neutrophils, 9% neutrophilic bands, 29% lymphocytes. Platelet count 254,000/mm³ (254 x 10⁹/L). Thyroxine (T4) and 12 panel serum chemistry including lactic dehydrogenase (LD), alkaline phosphatase, asparate aminotransferase (AST, SGOT), bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, uric acid, creatinine and cholesterol were within normal limits. Serum human chorionic gonadotropin (HCG) by a beta subunit assay was 130,000 miu/ml (maximum in normal pregnancy 50,000 miu/ml at 8-12 weeks). A chest film was normal. An obstetric ultrasound showed a uterus of about 20 weeks size with bizarre irregular echoes. No fetus was identified.

DR. PEDERSON: This patient's presentation is fairly typical for that of a hydatidiform mole (HM). This includes vaginal spotting, an elevated human

^{*} Resident in Family and Community Medicine, Sioux Falls, SD; Program affiliated with USD School of Medicine.

[†] Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Sioux Falls Faculty, USD School of Medicine, Sioux Falls, SD.

[‡] Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

chorionic gonadotropin (HCG) level, and an ultrasound showing bizarre, irregular echoes.

A HM is a benign abnormal product of pregnancy, which has remained in the uterus too long. The changes that take place are primarily in the trophoblast of the gestational sac. These include the following:

- Cystic degeneration which appears in the stroma of the villi with hydropic swelling in the main portions.
- 2. Lack of vascularization within the villi.
- 3. Despite the above, the villi remain viable and mild proliferation of the covering layers is noted. The functioning trophoblastic tissue produces excessive amounts of HCG and produces the positive pregnancy test. There is a loss of connection between the original fetal circulation and the villi. The villi begin to degenerate, pull apart, and become distended with fluid. With progressive swelling, grapelike masses are formed, thus producing the typical appearance of the HM.

The incidence of HM is approximately 1 in every 1500 pregnancies in the United States and Europe. In southeast Asia, or Taiwan specifically, the incidence increases to 1 in 125 pregnancies. There appears to be a recurrence rate of approximately 2% in a subsequent pregnancy which is a 4-5 times increased risk over a patient who has no previous history of HM. Molar pregnancies tend to be more common toward the beginning and the end of the childbearing years. Females over the age of 45 have a frequency 10 times that of the ages 20-40. The female presented today was in one of the lower risk groups as she was 23 years old and caucasian.

One of the most common findings in HM is vaginal spotting. 89% of the patients will present with vaginal bleeding ranging from spotting to profuse hemorrhage. This may occur just prior to expulsion, but more often occurs intermittently for weeks to months prior to the passage of the mole. Because of the blood loss, anemia is common. There may also be hemorrhage which is concealed within the uterus. The patient presented today had dark red vaginal bleeding at approximately 17 weeks gestation and also had bright red vaginal spotting from approximately 19-23 weeks gestation.

In over 50% of the cases, uterine size clearly exceeds the expected size for that duration of gestation due to rapid uterine enlargement seen characteristically in HM. Our case represented one of the 50% of cases which has a uterine size which does not exceed the expected size for that duration of gestation

Another finding which was not present in this case, was that of ovarian theca lutein cysts. These are present in approximately 30% of HMs and can be

fairly large. They may also increase in size for a short period after the mole has been evacuated. The theca lutein cysts, along with the trophoblast may cause an elevation in serum progesterone.

In this case, no fetal activity was noted, as expected in most cases of HM. Usually no fetal heart tones can be heard. Physical and ultrasound examination revealed no fetal heart tones or heart movements in this case. Rarely a twin gestation may occur in which an HM develops in one sac. Also, infrequently, there may be a molar change with a living fetus. In both of these cases, fetal heart tones would be able to be heard.

This patient had a normal blood pressure, but in 15% of the females with HM, hypertension may be seen. If pregnancy-induced hypertension is present, especially before the third trimester, a molar pregnancy needs to be considered. The syndrome of pregnancy-induced hypertension is rarely seen before 24 weeks. Therefore, if present before 24 weeks, a molar pregnancy deserves consideration.

Thyroxin levels (T4) may be increased, as in pregnancy, but hyperthyroidism is uncommon. In this case, the T4 was normal. One explanation for the rise in T4 is the TSH (thyrotropin) like activity of HCG.

Another "tip-off" for a molar pregnancy is the passage of vesicles spontaneously before the mole is aborted. This usually occurs at about 4 months gestation. It is rarely delayed beyond 7 months gestation. The findings of nausea, vomiting and abdominal cramps are also very common.

Trophoblast with or without villus stroma may escape from the uterus via the venous outflow in variable amounts. This can cause pulmonary vascular obstruction and death. Usually the volumes are small and the pulmonary symptoms are not noted.

HM or choriocarcinoma may produce metastatic pulmonary or occasionally hepatic or cerebral disease. The course is unpredictable. The metastases, left untreated, may regress spontaneously after evacuation of the uterus, may regress weeks or months later, or may cause the death of the patient. In this particular case, the chest x-ray was within normal limits.

If many of the characteristic physical findings are present, a molar pregnancy may be easily diagnosed. But if the typical findings are not present, it may be difficult to differentiate a HM from a pregnancy with uterine myomas, hydramnios, multiple fetuses, an error in menstrual data, or a normal pregnancy with a single fetus. Most of these other possibilities will demonstrate fetal heart tones or will show cardiac motion with an ultrasound examination.

The sonogram has the greatest diagnostic accuracy for HM. The typical ultrasound finding is that of

very irregular echoes from within the uterus. Rarely, a sonogram may be suggestive of HM in the following conditions:

- 1. Early pregnancy with uterine myoma.
- 2. Pregnancy with multiple fetuses.
- 3. Intrauterine death.
- 4. A tangential section of a normal placenta.
- 5. Early normal pregnancy.

In these instances, an ultrasound study may need to be repeated in a week or two along with a review of the course of the pregnancy.

Another test which may aid in the diagnosis of an HM is amniography. This is done by instilling intrauterine hypaque. This will show a honeycombed x-ray pattern. There is a 3% error in this technique and a slight risk of abortion. Sonography has now essentially replaced this technique.

Measurement of HCG levels is generally useful in the diagnosis of HM. The determination of serum levels is more accurate than urine levels. Characteristically the HCG level is found to be far above the normal range for the stage of pregnancy, so that a presumptive diagnosis of a molar pregnancy is made. In the first 2-3 months, very high levels may mean very little, as they are encountered occasionally in normal pregnancy, especially with multiple fetuses. But, beyond 100 days after the last menstrual period, there is a normal decrease in HCG, so that persistently high or increasing levels suggest abnormal trophoblastic growth. The HCG levels peak at approximately 65 days At 50,000 mIU/ml, then decrease to 15,000 to 30,000 mIU/ml for the remainder of pregnancy. If the HCG levels are well over the above ranges, a molar pregnancy is sug-

In summary, the supportive diagnostic features for a molar pregnancy include the following:

- 1. A uterus enlarged out of proportion for the duration of pregnancy (50%).
- 2. Inability to palpate fetal parts or see them on radiologic examination.
- 3. Presence of continuous or intermittent bloody vaginal discharge evident by approximately the 12th week of pregnancy. This is often more brown than red and usually is not profuse.
- 4. Elevated HCG levels in the serum according to the stage of pregnancy.
- 5. A typical sonographic pattern.
- 6. Pre-eclampsia or eclampsia earlier in pregnancy than is usually found.

All of the above findings were present in this case except the first and the last.

After the diagnosis of a molar pregnancy is made, therapy and follow up are of particular importance because of the possibility of aggressive disease. Approximately 80% of HMs will regress spon-

taneously. Fifteen percent will continue as nonmetastatic locally invasive gestational trophoblastic disease, and 5% will become metastatic gestational trophoblastic disease. The latter includes the subsequent occurrence of choriocarcinoma in about 2% of cases. It is this last 20% of the patients that are important to segregate and treat adequately, as their disease can be life-threatening. In 1939 there was a 1.4% mortality from HM. Now the mortality is close to 0.

The aggressive types of trophoblastic disease include invasive mole and choriocarcinoma. The invasive mole, often has excessive trophoblastic overgrowth with extensive penetration by the trophoblastic elements, including whole villi, into the depths of the myometrium sometimes to involve the peritoneum, adjacent parametrium, or vaginal vault. The invasive mole can only be distinguished from a more typical HM by noting these pathologic changes as histologic examination is unreliable.

Choriocarcinoma is a malignant tumor related to HM or invasive mole and characterized by the absence of a chorionic villi. It metastasizes very early to the lung (75%), vagina (50%), vulva, kidneys, liver, ovaries and brain. 40-50% of the choriocarcinoma follow a hydatidiform mole, 25-40% follow an abortion, 20-23% follow a normal pregnancy and it can rarely be co-existant with a normal pregnancy.

Therapy for HM includes immediate evacuation of the uterus and a continued follow up for the detection of any malignant changes. Vacuum aspiration is the best technique for evacuation of the mole. Oxytocin should be concommitantly administered to contract the uterus. Then gentle but sharp curettage is performed. With this technique, especially with larger moles, there is a risk of hemorrhage and perforation. Oxytocin, prostaglandin E2, or hypertonic saline techniques have been used in the past but are not recommended. If the female is older than 40, has 3 or more children, or has completed her childbearing, a hysterectomy may be considered. This will not eliminate metastatic trophoblastic disease, but does reduce the likelihood of its subsequent development. It should be remembered that females over the age of 40 are at the greatest risk for developing metastatic trophoblastic disease.

Prophylactic chemotherapy has been recommended by some, but probably should not be used because of the complications associated with evacuation of the mole (hemorrhage, infection, and uterine perforation) may necessitate a hysterectomy and the chemotherapeutic agents may contribute to mortality and morbidity. In addition, the long term effect of these agents is not completely understood.

Follow up is extremely important for the prompt detection of any changes suggesting metastatic or locally invasive trophoblastic disease. HCG levels

should fall progressively to undetectable levels. If the levels increase, then there is a proliferation of abnormal trophoblast or a normal pregnancy. Therefore, pregnancy needs to be prevented for adequate determination of the significance of the HCG levels.

To prevent pregnancy, oral contraceptive agents have been used but it has been observed that the need for chemotherapy for trophoblastic tumor in females who began oral contraceptives shortly after a HM evacuation was increased significantly. Also oral contraceptives cause a delay in the fall of the excretion of HCG. Therefore, an alternative birth control method may be preferred. An initial chest x-ray is important as a baseline.

The measures needed in follow up of HM include the following:

- 1. Measurement of serum HCG weekly (Beta Subunit HCG serum levels are preferred).
- 2. Prevention of pregnancy.
- 3. If chorionic gonadotropin levels continue to fall, withhold therapy.
- 4. When chorionic gonadotropin level is normal for 3 consecutive weeks, test monthly for 6 months.
- 5. After 6 months if there is no detectable serum HCG, follow up may be discontinued and pregnancy permitted. (Another source recommended repeat HCG levels every 2 months for an additional 6 months before discontinuing follow up and permitting pregnancy).
- 6. If the HCG level plateaus for more than 2 consecutive weeks, rises, or if metastases are detected, therapy for trophoblastic disease is necessary. This may include curettage or hysterectomy if there is no evidence of disease beyond the uterus. If the uterus is to be preserved (future pregnancies are desired) or there is evidence of lung lesions on chest x-ray, chemotherapy with or without curettage, is needed by agents which include methotrexate, actinomycin D, or a combination of the two.

To rule out extrauterine disease, the following are frequently required:

- 1. Physical examination.
- 2. Chest x-ray.
- 3. Intravenous pyelogram (IVP).
- 4. Liver and brain scan.
- 5. Liver and kidney function tests.
- 6. Pelvic ultrasound.
- 7. Selected CT Scans of the abdomen or pelvis.

If metastatic trophoblastic disease is found, careful follow up is again needed after treatment. The trophoblastic disease and chemotherapy do not appear to exert an adverse effect on subsequent pregnancies.

In summary, if trophoblastic disease is found, prompt therapy and follow up is necessary to prevent possible life-threatening sequelae.

Dr. Kim A. Pederson's Diagnosis Hydatidiform mole

Dr. Luechtefeld, will you please show the ultrasound examination.

DR. LUECHTEFELD: In our present case, no gestational sac nor evidence of a fetus were identified. Typical small cystic spaces scattered throughout an enlarged uterus were diagnostic of a molar pregnancy. No theca lutein cysts were identified. This is a classic picture of HM (Figure 1).

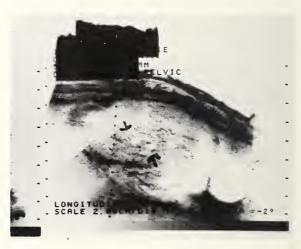


Figure 1 Ultrasound study. Arrows point to systic areas corresponding to hydropic villi.

In the diagnosis of hydatidiform mole by ultrasound, it is helpful to note that there is a roughly linear relationship between the gestational age of the molar pregnancy and the size of the vesicles. The cystic areas measure about 2 mm. in diameter at 8.5 weeks gestation, and about 10 mm. in diameter at 18.5 weeks. During the first trimester of the molar pregnancy, vesicles may be too small to be seen by ultrasound; sonographic findings may include only echogenic tissue within an enlarged uterus, and are often mistaken for a missed abortion. The pathognomonic sonographic findings of the second trimester HM include an enlarged uterus containing low to moderate amplitude echoes with numerous small fluid-containing spaces scattered throughout. As mentioned previously, the uterine enlargement may be disproportionately greater than expected for gestational age, although this was not the case in the patient under discussion. The uniform distribution of the vesicular pattern on ultrasound is quite helpful in distinguishing the HM from a missed abortion or a degenerating leiomyoma. Also, about one-third of

cases of molar pregnancies will have bilateral theca lutein cysts which can be demonstrated by ultrasound. They were not seen in this case.

On performing the ultrasound examination, one should be very careful to determine whether the vesicles are diffuse or focal, whether any normal placental tissue can be identified, and whether a fetus is present or not. If the vesicles are focally distributed, or if non-cystic normal placental tissue is identified along with cystic placenta, then the diagnosis is more probably partial mole, hydropic degeneration of the placenta, or missed abortion.

Hydropic degeneration of the placenta may be extremely difficult to distinguish from molar tissue by ultrasound, but generally the vesicles are less extensive in the former. In a missed abortion, the cystic areas are not uniformly spread, they are often found in only one portion of the uterus rather than diffusely, the uterine size should be normal or small for dates, a distorted gestational sac may be seen, and background echoes surrounding the cystic area will be coarse and clumped rather than diffusely spread as seen with the molar pregnancy. In cystic degeneration of a leiomyoma, one would search for nodular enlargement of the uterine outline and would try to demonstrate the highly attenuating properties of the fibroid.

The sonographic appearance of the invasive mole is not specific. Well circumscribed echogenic spherical masses may be seen within the wall of the uterus; the enlarged uterus may show areas of irregular sonolucency secondary to hemorrhage and necrosis of the myometrium, but one may not be able to distinguish these features from other uterine tumors.

In the evaluation for metastatic disease, ultrasound may be helpful in determining the extent of the pelvic tumor mass, in demonstrating the amount of necrotic and hemorrhagic changes in the tumor mass, in identifying obstructive hydronephrosis, and in evaluating the liver for hepatic metastases.

If a subsequent rise in the beta HCG is seen after evacuating a hydatidiform mole from the uterus, ultrasound will be most helpful in distinguishing between a recurrent mole and a normal intrauterine pregnancy.

DR. BARLOW: Suction and sharp curettage of the endometrial cavity was performed by Dr. Dean Madison*. We received an aggregate of clear edematous gray to tan structures measuring in mass 29 x 25 x 2.5 cm. typical of a HM (Figure 2). On microscopic examination, the changes described by Dr. Pederson were present. They were large edematous villi with central cavitation surrounded by areas of



Figure 2
Gross picture of hydatidiform mole with many hydropic villi.

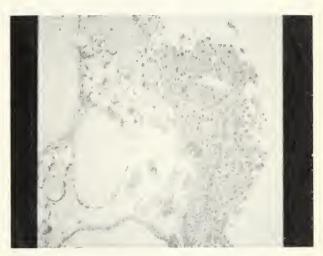


Figure 3 Trophoblastic proliferation about pale, edematous villi on microscopic examination H&E $100 \times$

prominent tropoblastic perforation (Figure 3). I would like to emphasize that the clinical and sono-graphic examination and gross appearance are important in the diagnosis of an HM. Hydropic villi are not uncommon in spontaneous abortion. However, the clinical, ultrasonic and gross pathologic changes accompanied by the classic microscopic features described above, make the diagnosis of a typical HM.

FINAL ANATOMIC DIAGNOSIS HYDATIDIFORM MOLE

Pathologists have tried for many years to predict the outcome of HM on the basis of the extent and dysplastic qualities of the tropoblastic proliferation. The more extensive and atypical the trophoblastic proliferation, the more likely it is that invasive mole

^{*} Obstetrician and gynecologist, Sioux Valley Hospital Faculty, USD School of Medicine, Sioux Falls, S.D.

or metastatic disease would be a sequela. However, there are many exceptions to the rule. Close microscopic examination not only of the tissue passed per vaginam, but of the curetted tissue adjacent to the myometrium is necessary before accurate prediction can be attempted. No matter what the pathologic diagnosis is, the patient requires follow up as Dr. Pederson has outlined.

DR. DEAN MADISON: Follow up of this patient has indicated a progressive decline in the HCG levels. There has been no evidence of invasive or metastatic disease.

DR. DANIEL HEINEMANN*: If a patient had a missed abortion, would there continue to be detectable HCG?

DR. BARLOW: Yes, as long as trophoblastic tissue is present in the body, sensitive beta subunit assays may detect HCG.

It is important to realize that there are several types of pregnancy tests. Immunologic tests employing an antibody to the whole HCG molecule are notoriously insensitive and imprecise. Antibodies directed toward the beta subunit portion of the HCG molecule are better but vary in both sensitivity and precision. One modification is the radioreceptor assay which utilizes cow corpus luteum as a receptor. This test is very sensitive but does give false positive tests due to crossreactions with luteinizing hormone (LH) in perimenopausal women and at the time of the LH surge in females in the reproductive age group. The radioimmunoassay is the most sensitive and precise method for pregnancy testing and can be done as a qualitative or quantitative measurement. The former can be used as a test available in three hours to diagnose early or abnormal pregnancy such as ectopic pregnancy. The latter is performed by using an overnight incubation and is the method of choice for monitoring the serum HCG levels after evacuation of a molar pregnancy.

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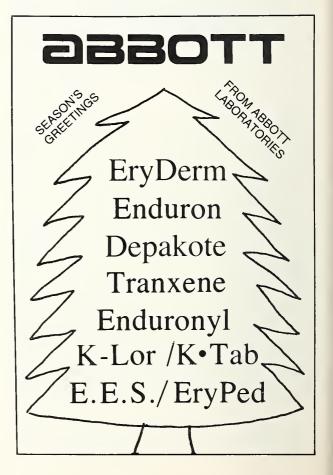
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^{*} Resident in Family Community Medicine, Sioux Falls, SD.

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4:00 p.m. SDAFP Board of Directors Meeting

THURSDAY, FEBRUARY 2, 1984

MORNING SESSION

L. W. Finney, M.D., Moderator

7:00-7:30 a.m. Registration

Complimentary continental breakfast 7:30-8:10 a.m. Case #1: Rheumatoid Arthritis

Arthritis of the Hands in a Young Woman

Alan L. Rosenberg, M.D. Case #2: Osteoarthritis

8:15-8:55 a.m. Chronic Knee Pain in a 46-Year-Old Man

Herbert Kaplan, M.D.

9:00-9:40 a.m. Panel on morning topics 9:45-5:30 p.m. WINTER SPORTS TIME

EVENING SESSION

Charles Swanson, M.D., Moderator

5:00-5:30 p.m. Registration

Complimentary coffee, hot wine and hot

buttered rum

5:30-6:10 p.m. Case #3: Hyperuricemia and Gout

Acute Arthritis of the Knee in a 37-Year-

Old Man

Alan L. Rosenberg, M.D. Case #4: Ankylosing Spondylitis 6:15-6:55 p.m.

Low Back Pain in a Young Man

Herbert Kaplan, M.D.

7:00-7:45 p.m. Panel on evening topics

7:45 p.m. **EVENING FREE**

FRIDAY, FEBRUARY 3, 1984

MORNING SESSION

Michael Brown, M.D., Moderator

7:00-7:30 a.m. Registration

Complimentary continental breakfast

7:30-8:10 a.m. Pulmonary Function Tests and Arterial

Blood Gases Donald Pell, M.D.

8:15-8:55 a.m. Non-Articular Rheumatism - I

Gary Ruoff, M.D.

9:00-9:40 a.m. Non-ARDS Respiratory Failure Donald Pell, M.D.

9:45-5:30 p.m. WINTER SPORTS TIME

EVENING SESSION

Richard Finley, M.D., Moderator

5:00-5:30 p.m. Registration

7:00-7:40 p.m.

Complimentary coffee, hot wine, hot but-

tered rum

Asthma: Evaluation & Therapy 5:30-6:10 p.m.

Donald Pell, M.D.

Non-Articular Rheumatism - II 6:15-6:55 p.m. Gary Ruoff, M.D.

Ventilator Care and Tube Management

Donald Pell, M.D. 7:45-8:15 p.m. Panel with Friday speakers

8:15 p.m. **EVENING FREE**

SATURDAY, FEBRUARY 4, 1984

MORNING SESSION

Richard W. Honke, II, M.D., Moderator

7:00-7:30 a.m. Registration

Complimentary continental breakfast 7:30-8:10 a.m. Adult Respiratory Distress Syndrome/

Shock Lung

William J. Howard, M.D.

8:15-8:55 a.m. Chronic Pain in Family Practice

Gary Ruoff, M.D.

9:00-9:40 a.m. Hemoptysis

William J. Howard, M.D.

9:45-10:15 a.m. Panel of morning speakers 10:15 a.m. SEMINAR CLOSES

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will also affect duration. ¹⁸ The loss of effect in the C.S.F. is due to clearance. The more lipid soluble the drug, the quicker it is cleared. ¹⁶

After the initial reports in animal studies and man. volunteer studies on pain-free humans were carried out by Bromage to determine their effects. In one study injection of methadone and dilaudid into the thoracic and lumbar epidural space caused intense segmental analgesia with minimal respiratory depression or urinary retention. 19 When Bromage compared 10 mg. morphine administered intravenously versus the lumbar epidural route, he found greater analgesia and more side effects in the epidural route. Analgesia was initially confined to the lower extremity and after 3-4 hours spread cephalad to involve the upper extremities and even the face in 50% of the subjects. This lasted 16-22 hours.²⁰ Samii, with epidural morphine, found pruritus occurring at 3 hours, nausea at 4 hours, and vomiting at 6 hours. As with respiratory depression the above occurred in a dose dependent manner and could also be reversed with naloxone in a dose dependent manner.21

There are two stages of respiratory depression with morphine. The early stage is usually seen within one hour and is secondary to vascular uptake from the epidural space. This stage would not likely be seen with intrathecal administration (Figure 1). The late stage, which can be seen with both routes, is

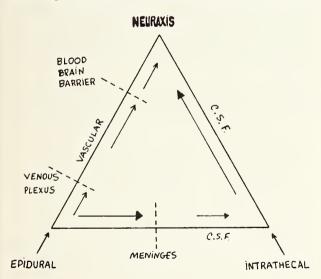


Figure 1
Schematic diagram demonstrating relationship between epidural and intrathecal injections. The epidural injection can result in some absorption into the blood through the internal vertebral venous plexus possibly causing an early stage of respiratory depression. Parenteral narcotics must bypass the blood-brain barrier to be effective. Most of the epidural injection will enter the cerebrospinal fluid through the dural root sleeves and exert its effect on the neuraxis. The intrathecal injection directly enters the cerebrospinal fluid and exerts its effect. Late respiratory depression can occur because of cephalad spread.

related to the lipid/water solubility of the narcotic and to cephalad spread by C.S.F. flow within the subarachnoid and ventricular systems. Respiratory depression is greatest 6-10 hours after injection but may be seen as late as 23 hours. Naloxone will reverse the depression, but repeated doses probably will be necessary. ^{22, 23, 24} Present evidence suggests that the lipid soluble narcotics methadone, meperidine, and fentanyl diffuse from the C.S.F. to spinal cord lipids rapid enough to probably avoid the cephalad spread seen with the poorly soluble morphine. ¹⁸

Intraspinal (i.e. epidural and intrathecal) narcotics are being used in the treatment of acute (postoperative, obstetric, post-traumatic) and chronic (cancer, other debilitating illness) pain. 11, 16 Bromage, in a recent review, summarized the results of many studies on the use of these techniques. Analgesia is dose dependent and initially segmental. Larger doses cause greater analgesia and duration. When fentanyl (highly lipid soluble) is injected into the epidural space, onset is rapid, distribution is segmental, and duration is short. Morphine (poorly lipid soluble) injection, however, results in slow onset, more general distribution, and prolonged duration (16-24 hours vs. 1-2 hours for fentanyl). 18 Other narcotics have intermediate durations of action (Table I). Serum levels following epidural in-

The doses administered in	Table and durations 10 cc. balanc epidural sp	of narcotics ed salt soluti	
Drug	Lipid Sol	Dose	Analgesia
Fentanyl Levodromeran Meperidine Morphine	high moderate moderate low	0.1 mg. 4 mg. 100 mg. 5 mg. 10 mg.	6 hrs. 9 hrs.

jections of morphine closely parallel those of intramuscular injections. The analgesic effect following epidural morphine, however, correlates poorly with serum morphine levels and remains profound for 18 hours to several days. 25 Chronic pain is easier to control than acute pain. Smaller doses of narcotics can be used. Intrathecal morphine can control labor pains whereas the epidural route is not as effective. Generally, 1-2 mg. morphine intrathecally can relieve visceral pain during the first stage of labor without any effect on established labor or newborn. However, if repeat injections were necessary during the second stage, an epidural approach with a catheter in place would be more practical. 11

In order to minimize the occurrence of respiratory depression, dosage should be restricted to the minimal amount necessary for analgesia. ²¹ Injecting

It is important to note that when narcotics are injected into the intrathecal and epidural compartments, the preservative-free form of the drug must be used.

as close to the dermatone level of pain one wishes to control and placing the patient in a head-up tilt while injecting would also be helpful. 12, 21 Because respiratory depression is inversely related to the length of the vertebral column, it would be less likely in taller patients for comparable doses. 26

Generally, the intrathecal route would be best when the narcotic could be combined with a spinal anesthetic or only a single injection would be necessary (as diagnostic relief of chronic or cancer pain to determine the suitability of a catheter implantation or post-operative pain relief when injected at the end of a surgical procedure with the patient anesthetized). Prolonged administration of intraspinal narcotics is done through an insertion of an indwelling catheter. The catheter can be safely employed through either extradural or intrathecal approaches. A permanent intrathecal catheter can be extended subcutaneously, lateral from the subarachnoid space, and be connected to a reservoir (e.g., Cordis drug infusion reservoir) (Figure 2). The reservoir is commonly placed subcutaneously subcostally about the level of the anterior axillary line, and its location allows for easy injection by nursing personnel or family members at home.²⁷ One-half to two mg. doses of morphine are injected Q8-12 hours. Smaller' doses, as given with the intrathecal approach, appear to limit respiratory depression and more frequent injections seem to result in less development of tolerance. 26, 28

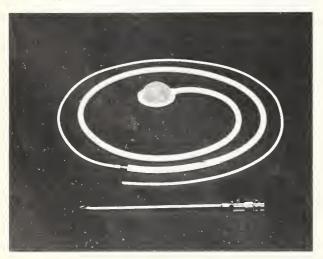


Figure 2
The Cordis drug infusion reservoir used for chronic intrathecal narcotic injections. (Drug infusion reservoir. Number 9EO458 Photograph courtesy of Cordis Corporation, Miami, Florida, 33152).

The epidural route can also be used. For postoperative pain, a standard epidural catheter can be used and left in place for several days, if necessary. Dannemiller recommends the use of the wire-wound Racz catheter for epidural insertion since it is easy to handle and unlikely to kink.²⁸ In addition, it is designed to be used without a removable stylet, has a "spring like" tip to prevent inadvertent dural puncture, and is designed to be left in the epidural space for extended periods of time.²⁹ Although a definite advantage in the treatment of long term pain, there was a recent report in some patients of difficulty removing these catheters. 30 Malignancy pain can be controlled with smaller doses of morphine in the epidural space such as 2-4 mg. Postoperative or acute pain may require 6-8 mg. morphine. It is important to note that when narcotics are injected into the intrathecal and epidural compartments, the preservative-free form of the drug must be used. Preservatives, such as phenol, sodium formaldehlyde sulfoxylate, and metacresol, could be neurotoxic. Although tubex syringes are classified as "preservative-free," these should not be used for intraspinal injections. 31 Techniques involving intermittent injections and continuous infusions of narcotics through an implanted reservoir for chronic treatment have been studied. 32, 33 The use of a volumetric infusion pump has been found to be very helpful and can eliminate the inconvenience of reinjecting over a long period of time.³⁴ It may well be that the acute pain or trauma and surgery will, in the future, be best controlled with a continuous infusion of a lipid soluble narcotic. 14

The key to safe management of these patients lies with proper monitoring of vital signs, most importantly respiration.

In the hospital, these patients should be monitored in an "intensive care-like" setting post-injection. 18, 27 With many major surgical cases such as thoracotomies, aortobifemoral bypasses, multisystem trauma, etc., this would not be a problem since they would ordinarily be admitted to the Intensive Care Unit (ICU). Other surgical, as well as chronic pain, patients, however, might not otherwise have to be admitted to the I.C.U. Here an intermediate care unit (such as post-coronary) would be helpful and would save I.C.U. space for the more critically ill patients. Once a proper dose regimen is established in these patients and their vital signs have remained stable without any evidence of respiratory depression, they are transferred to their referring ward or home. On the average, this requires 24-72 hours. Nursing personnel, responsible It is important to note that the use of intraspinal narcotics should not be accompanied by parenteral narcotic administration.

family members, and patients are instructed in proper injection technique as well as potential problems and treatment. When a narcotic is being infused continuously into the epidural space, the patient should remain in the intermediate care setting as long as the infusion is running.¹⁸

The key to **safe management** of these patients lies with proper monitoring of vital signs, most importantly respiration. Respiratory rate is a poor index of depression and apneic episodes may occur with little warning. Apneic monitors have not been found to be helpful or reliable in these patients.³⁵ A standing order should be written so that when respiratory depression or other side effects are present, naloxone should be administered immediately intravenously by the nurse (0.1-0.2 mg. dose). It is important to note that the use of intraspinal narcotics should **not** be accompanied by parenteral narcotic administration.¹²

The use of intraspinal naracotics is revolutionary and has many possible future benefits. It can provide pain relief of longer duration with smaller doses in patients having acute and chronic pain previously resistent to other treatment. Intraspinal narcotics are a mode of treatment to be considered along with the other modalities previously mentioned but not necessarily replacing them. It remains for us in anesthesiology and other specialties interested in pain management to determine which patients can be properly controlled by a specific method and when more comprehensive data is available to make specific recommendations.

We still do not have all the answers to many questions on intraspinal narcotics. Hopefully, however, the above information has provided insight into a promising area of pain control and will be useful in determining which patients might benefit from this new mode of pain therapy. In conclusion, Bromage has stated regarding this technique: "Accumulating evidence suggests that intelligent manipulation and exploitation of relevant physicochemical and pharmacological variables will lead to the development of rational clinical regimen that will be both safe as well as more effective and controllable than other analgesic options." 12

ACKNOWLEDGMENT

The author would like to express his appreciation to Richard G. Belatti, M.D. and William J. Horner, M.D., for their critique of the article and to John Olson, Sioux Valley Hospital, for his photography.

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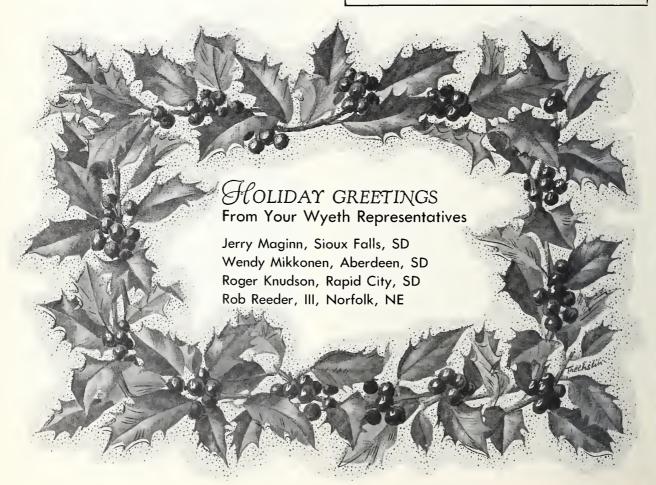


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S Auxiliary News



A Day at The Capitol

Dear Physician!

YOU AND YOUR SPOUSE ARE INVITED TO A "DAY AT THE CAPITOL" ON TUESDAY, JANUARY 24, 1984 AT THE HOLIDAY HAUS, PIERRE, SD. COCKTAILS AND DINNER AT 6:30 PM.

Please contact your legislator(s) and invite them to join you, and our group. They will be **your** guests. Be sure to give them exact details on date, time and location.

The Medical Society membership needs to make larger strides in legislative efforts and political action. The Auxiliary must continue to play a role to maximize our effectiveness in the Legislature. Every member's efforts are important and appreciated. However, to assure success, our legislative effort (communicating with legislators on issues) and political action (contributions of time, money and energy needed to elect candidates) must multiply.

The key to countering the trend toward enactment of legislation **not** in the best interest of the public and South Dakota medicine, is to improve and expand relationships between South Dakota lawmakers and their constituent medical families.

You can help in a number of ways. GET IN-VOLVED in local political party activities. WORK in political campaigns. BECOME FAMILIAR with medically-related legislative issues. DISCUSS political and legislative issues affecting medicine with spouses. BECOME MEMBERS of the South Dakota Political Action Committee (SoDaPAC) and the American Medical Political Action Committee (AMPAC).

If you still don't feel inspired to get involved, then keep in mind what one wise old man from Greece said, "THE PUNISHMENT FOR WISE MEN WHO REFUSE TO TAKE PART IN THE AFFAIRS OF GOVERNMENT — IS TO LIVE UNDER THE GOVERNMENT OF UNWISE MEN."

MERRY CHRISTMAS AND HAPPY NEW YEAR TO YOU FROM THE SDSMA AUXILIARY!!

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Marie Hovland, President South Dakota State Medical Auxiliary

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Apr.—p. 17, May—p. 23, June—p. 13,			Nitrous Oxide, a Health Hazard??	
Aug.—p. 30, Sept.—p. 13, Oct.—p. 21,			John B. Gregg, M.D. July	20
Nov.—p. 15, Dec.—p. 14 Pulmonary Embolus With a Normal Ventilation			Is a Statewide Tumor Registry Needed for South	
Perfusion Lung Scan: Case Report			Dakota? John B. Gregg, M.D.	
Leonard M. Gutnik, M.D.	July	17	Lawrence J. Massa Sept.	14
Q			Fetal Alcohol Syndrome — Intrauterine Child	
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Thirty Year Old Caucasian Female With Abdominal Pain of 11 Days Duration			Tam, Guy E., M.D. Myxoid Tumor of the Uterus and Right Atrial	
Delwin K. Ohrt, M.D.			Myxomas	
Richard A. Jaqua, M.D.	Oct.	25	John F. Barlow, M.D.	
Reaction Paper — Becoming a Doctor			Samir Abu-Gazeleh, M.D.	
John M. Rud, MSII Roberts, Kim Meyer, B.A.	Feb.	13	Patricia S. Wirtz, M.D.	
Ear Disease and Hearing Loss, Pierre, South			Lewis C. Ofstein, M.D. Charles P. O'Brien, M.D.	
Dakota, 1962-1982			Gail L. Woods, M.D.	
John B. Gregg, M.D.			Walter G. Drymalski, M.D. July	9
Michael J. Colleran, M.A., C.C.C.	Oct.	9	This Is Your Medical Association	
Role of Tracheostomy in Acute Laryngeal Obstruction in Children (The)	cuon		Feb.—p. 27, May—p. 33, June—p. 26, July—p. 21, Sept.—p. 27, Oct.—p. 20, Nov.—p. 16,	
Thomas G. Bunker, M.D.			Dec.—p. 27, Oct.—p. 20, Nov.—p. 10,	
Winston B. Odland, M.D.	June	5	Transactions Of The South Dakota State Medical	
D District	Aug.	35	Association 102nd Annual Meeting Aug.	5
Roster — District Rud, John M., MS11	Aug.	31	U	
TO 1 TO 1	Feb.	13	Ulrich, Robert C., M.D.	
Rural Family Medicine Clerkship (RFMC) at the			Clinicopathological Conference	

Month Page Fifty-Six Year Old Caucasian Female With Infection of Knee and Acute Renal Failure Richard A. Jaqua, M.D. John F. Barlow, M.D. Nov. 5 Van Denmark, Robert, M.D. Simple Method of Treatment of Fractures of the Fifth Metacarpal Neck and Distal Shaft (Boxer's 5 Fracture) (A) Wirtz, Patricia S., M.D. Myxoid Tumor of the Uterus and Right Atrial Myxomas John F. Barlow, M.D. Samir Abu-Gazeleh, M.D. Guy E. Tam, M.D. Lewis C. Ofstein, M.D. Charles P. O'Brien, M.D. Gail L. Woods, M.D. Walter G. Drymalski, M.D. July Woods, Gail L., M.D. Myxoid Tumor of the Uterus and Right Atrial Myxomas John F. Barlow, M.D. Samir Abu-Gazeleh, M.D. Guy E. Tam, M.D. Patricia S. Wirtz, M.D. Lewis C. Ofstein, M.D. Charles P. O'Brien, M.D. Walter G. Drymalski, M.D. July \mathbf{X} Y

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S Future Meetings

January

- Hawkeye Sports Medicine Symposium, Iowa Mem. Union, U. of Iowa, Iowa City, IA, Jan. 5-7. Contact: Dir. of Conf., Ctr. for Conf. & Instit., Iowa Mem. Union, Iowa City, IA 52242. Phone: (319) 353-5505.
- 15th Annual Cardiovascular Conference at Snowmass, Snowmass Resort, Snowmass, CO, Jan. 16-20. Fee: \$295 mem; \$345 nonmem. 20 hrs. AMA Category I credits. Contact: Am. Coll. of Card., Ronald J. Sanchez, Dir. of Comm., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.
- Cardiac Imaging Seminar, Prospector Square Hotel, Park City, UT, Jan. 29-Feb. 3. Fee: \$325 mem; \$400 nonmem. 24 hrs. AMA Category I credits. Contact: Am. Coll. of Card., Ronald J. Sanchez, Dir. of Comm., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.

February

- Current Concepts in Perinatal Medicine, Radisson Plaza Hotel, St. Paul, MN, Feb. 16-17. AMA Category I credits. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Consultative Cardiology: Update in Diagnostic and Therapeutic Techniques, Marriott Hotel, Newport Beach, CA, Feb. 16-18. Fee: \$250 mem; \$300 nonmem. 15 hrs. AMA Category I credits. Contact: Am. Coll. of Card., Ronald J. Sanchez, Dir. of Comm., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.

March

- Family Practice Update, St. Joseph's Hosp., St. Paul, MN, Mar. 2-3. 10 hrs. AMA Category I credits. Contact: Charles W. Drage, M.D., Dir. CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.
- Fifth Annual Mammoth Mountain Emergency Medicine Ski Conference, Mammoth Lakes, CA, Mar. 4-9. Fee: \$325. 20 hrs. AAFP & AMA Category I credits. Contact: Daniel Abbott, M.D., Prog. Dir. Med. Conf., P.O. Box 52-B, Newport Beach, CA 92662. Phone: (714) 650-4156.
- Refresher Course for the Family Physician, U. of Iowa, Iowa City, IA, Mar. 6-9. Contact: Richard Caplan, M.D., Asso. Dean for CME. U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, Mar. 7. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Current Concepts in Cardiopulmonary Medicine, Radisson Plaza Hotel, St. Paul, MN, Mar. 8-10. AMA Category I credits. Contact: St. Paul-Ramsey Mcd. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 22I-3992.
- Occupational and Environmental Pulmonary Diseases, Radisson Plaza Hotel, St. Paul, MN, Mar. 10. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

- Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, Mar. 15. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Review and Update in General Pediatrics and Family Practice, Princess Hotel, Freeport, Grand Bahamas, Mar. 18-25. Fee: \$350. 20 hrs. AAFP & AMA Category I credits. Contact: Marge Adey, U. of Neb. Med. Ctr., 42nd & Dewey, Omaha, NE 68105. Phone: (402) 559-4152.
- Otolaryngology Clinical Conference, U. of Iowa, Iowa City, IA, Mar. 23. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Iowa-Western Illinois Neurological Association: Pediatric Neurology, Mar. 23-24. Contact: Richard Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.
- Biofeedback in Perspective: Fifteen Years of Development, Regent Hotel, Albuquerque, NM, Mar. 23-28. Contact: Biofeedback Soc. of Am., 4301 Owens St., Wheat Ridge, CO 80033. Phone: (303) 422-8436.
- Obstetrics Update, St. Paul Hotel, St. Paul, MN, Mar. 24-25. 14 hrs. AMA Category I credits. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

March

St. Moritz 1984: Advances in Diagnostic Imaging, Palace Hotel, St. Moritz, Switzerland, Mar. 24-Apr. 1. 20 hrs. Category I credits. Contact: Educational Symposia, P. O. Box 17241, Tampa, FL 33682. Phone: (813) 971-6000, ext. 1112.

April

International Breast Cancer Conference, Tianjin, China, Apr. 24-27. Contact: Mr. C. H. Tu, Manager Dir., Inter-World Exchange Service, 401/2 & 404/5 Metropole Bldg., 57 Peking Rd., Kowloon, Hong Kong, China.

Minnesota Medical Association Resource Group on Rheumatic Diseases

Presents

RHEUMATOLOGY SEMINAR V March 6-March 13, 1984

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CONTACT: Department of CME and Meeting Services,

Minnesota Medical Association, Suite 400, 2221 University Avenue SE, Minneapolis,

Minnesota 55414, 612/378-1875.





